ARCHIVES OF OTOLARYNGOLOGY

VOLUME 37 MAY 1943 NUMBER 5

COPYRIGHT, 1943, BY THE AMERICAN MEDICAL ASSOCIATION

HISTOPATHOLOGIC CONSIDERATIONS IN TREATMENT OF THE EUSTACHIAN TUBE

CAPTAIN J. BROWN FARRIOR

MEDICAL CORPS, ARMY OF THE UNITED STATES, UNIVERSITY OF MICHIGAN AFFILIATED UNIT

Developments in otology in the past several years have focused increasing attention on the eustachian tube and its relationship to the disease processes within the cavity of the middle ear. The reports of Crowe and Burman [1] on the use of irradiation of the pharyngeal orifice of the eustachian tube in the treatment of deafness have aroused new interests in the role of changes in the eustachian tube as the pathogenic factor. Studies in aero-otitis-media by Armstrong and Heim [2] have glorified the physiology of the eustachian tube and have presented a new otologic entity which is dependent on disturbed physiologic activity. Perlman [3] has presented a quantitative method of study of the patency of the eustachian tube.

Whether the clinical manifestation is conductive deafness, catarrhal otitis media or aero-otitis-media, the primary factor in disease of the middle ear is almost invariably some histopathologic process within the eustachian tube itself. The histopathologic changes in the eustachian tube, except for rare neoplasm, are those of inflammation. Knowledge of the histopathology of inflammation is complete, from the primary vascular response to repair by the proliferation of fibroblasts and the formation of cicatricial tissue. Failure to consider this histopathologic process has, in the past, led to the abusive employment of mechanical therapeutic measures, which tend not to promote the resolution of the disease process but to encourage the formation of scar tissue. The cicatricial tissue thus produced has in many cases impaired the function of the eustachian tube, with resultant disease of the middle ear. By correlation of knowledge of the existing histopathologic changes with the clinical manifestations of the disease, the problem of therapy can be logically approached. The therapeutic conclusions reached by such a rationale are thereby based on indisputable histopathologic knowledge.

The basis of the study is the microscopic examination of 100 histologic sections of eustachian tubes removed at autopsy. The entire eustachian tube and the adjacent portion of the nasopharynx were removed through the cranial cavity. In each case the right eustachian tube was sectioned transversely at intervals of 4 mm. and the left eustachian tube longitudinally. The specimens removed included normal eustachian tubes, as well as tissue revealing the respective inflammatory processes. One specimen was taken from a 4 year old boy who died eight hours

From the Department of Otolaryngology, University of Michigan Medical School.

1. Crowe, S. J., and Burman, C. F.: Recognition, Treatment and Prevention of Hearing Impairment of Children, Ann. Otol., Rhin. & Laryng. **50**:15-31 (March) 1941.

2. Armstrong, H. G., and Heim, J. W.: The Effect of Flight on the Middle Ear, J. A. M. A. **109**:417-421 (Aug. 7) 1937.

3. Perlman, H. B.: The Eustachian Tube: Abnormal Patency and Normal Physiologic State, Arch. Otolaryng. **30**:212-238 (Aug.) 1939.

after mastoidectomy and adenoidectomy. Another specimen was obtained from a patient who died of an intracranial pathologic condition twenty-four hours after catheterization of the left eustachian tube for tubotympanitis.

HISTOPATHOLOGIC CONSIDERATIONS

Within the eustachian tube infection, allergy and trauma are the irritants which most frequently incite the inflammatory process. Although the process of inflammation is continuous from the time of injury until complete healing has taken place, it is usually divided into two phases: first, the phase of vascular and cellular response, the inflammatory phase; and, second, the phase of healing, or the phase of repair. The inflammatory processes within the eustachian tube manifest themselves histopathologically by changes in the epithelium, the subepithelial connective tissue, the mucous glands and the lymphoid tissue. Normally the eustachian tube

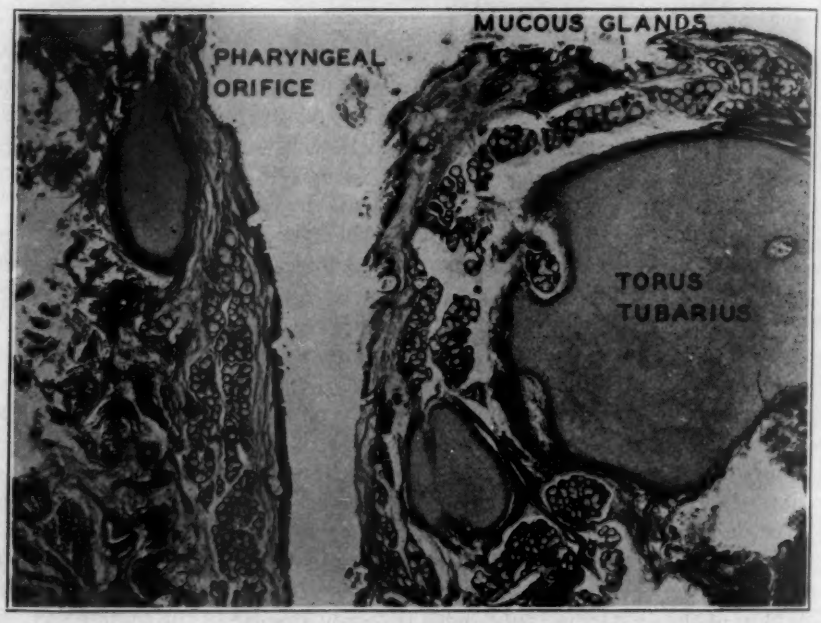


Fig. 1.—The normal pharyngeal orifice of the eustachian tube. The mucous membrane is pseudostratified columnar epithelium. The stroma is divided into a loose subepithelial layer and a dense perichondrial or periosteal layer. The subepithelial layer, containing numerous mucous glands, is subject to the greatest change during inflammation.

is lined by pseudostratified, ciliated columnar epithelium, in which the goblet cells are secreting mucus (fig. 1). The first change in the epithelium during an inflammatory process is an increase in mucin within the goblet cells and an increase in the secretion of mucus; second, the cilia and epithelium may be entirely destroyed by a suppurative process. The stroma of the eustachian tube consists of a subepithelial layer of loose areolar connective tissue and a perichondrial or periosteal layer of dense fibrous connective tissue. The inflammatory response is largely in the subepithelial layer of areolar connective tissue, which contains the mucous glands and the lymphoid nodules. In an inflammatory process there are edema and cellular infiltration in this subepithelial tissue. If the inflammatory reaction progresses to suppuration, there is actual necrosis in the subepithelial layer. After every inflammatory reaction repair takes place by the absorption of the tissue fluids, cellular exudate and fibrin, and, as a part of this healing process,

there are the formation of new blood vessels and the proliferation of fibroblasts in the subepithelial tissue. Numerous mucous glands are present in the stroma of the eustachian tube. These glands are most abundant inferiorly about the pharyngeal orifice and decrease in number toward the middle ear (fig. 1). Normally the individual acini of these glands are in various stages of secretory activity. However, during an inflammatory process the cells in all the acini secrete mucus at their maximal rate of production (fig. 2). If there is mechanical obstruction to the flow of mucus by inflammatory edema, lymphoid tissue or scar tissue, the mucus may be retained in dilated acini or ducts.

There is no lymphoid tissue in or about the normal eustachian tube. However, with chronic disease of the nose, nasopharynx and eustachian tube, lymphoid nodules may appear anywhere in the subepithelial tissue (fig. 3). These nodules are most frequently observed in the inferior aspect of the pharyngeal orifice of

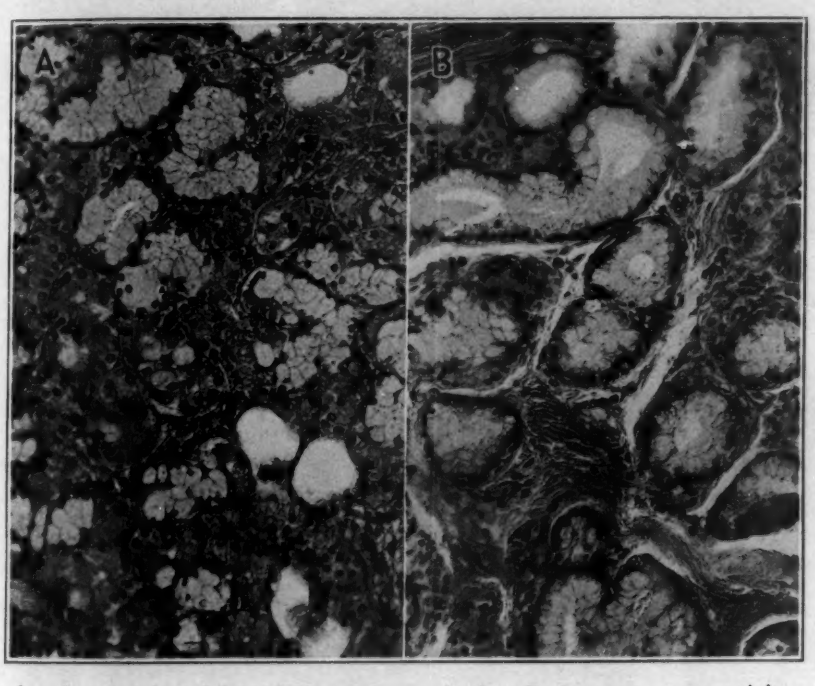


Fig. 2.—*A*, normal activity of the mucous glands; *B*, hypersecretory activity of the glands in inflammation.

the tube and over the torus tubarius. However, they have been noted high within the lumen of the tube and even within the middle ear itself. These lymphoid nodules may act as a mechanical factor in obstructing the lumen of the eustachian tube.

The inflammatory process within the eustachian tube may be acute catarrhal, acute purulent, acute suppurative, chronic catarrhal or the result of trauma.

Acute Catarrhal Inflammation.—Acute catarrhal inflammation of the eustachian tube is the mild inflammatory process which is seen in cases of acute infection of the upper respiratory tract or of allergic rhinitis. Its chief characteristic is the great increase in the production of mucus. There are edema of the subepithelial tissues and slight cellular infiltration. Microscopically (fig. 4) the goblet cells are filled with mucus. Leukocytes are seen passing through the epithelium. However, there is no destruction of cilia or of the continuity of the epithelium. In the subepithelial connective tissue the vessels are dilated; there are pronounced

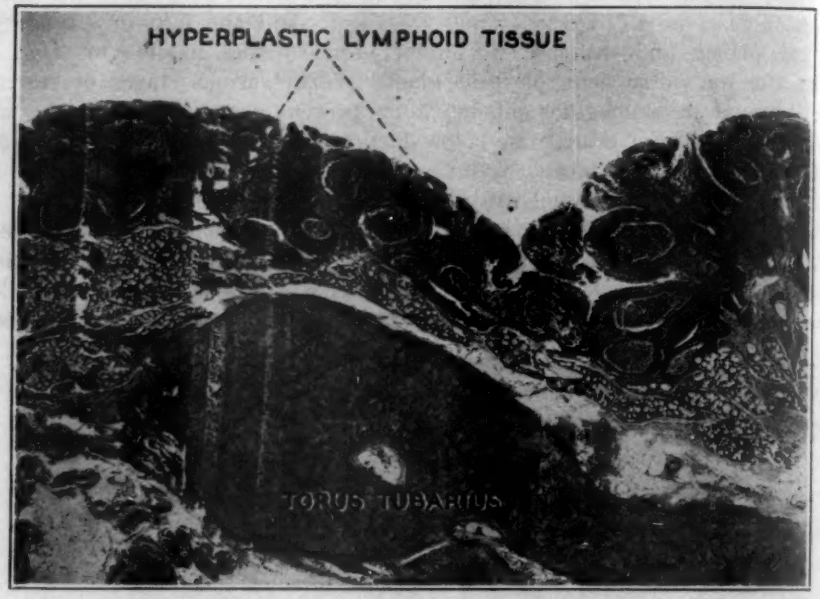


Fig. 3.—Inferior portion of the torus tubarius, revealing pronounced hyperplasia of the lymphoid tissue about the pharyngeal orifice of the eustachian tube in a child aged 7 years.

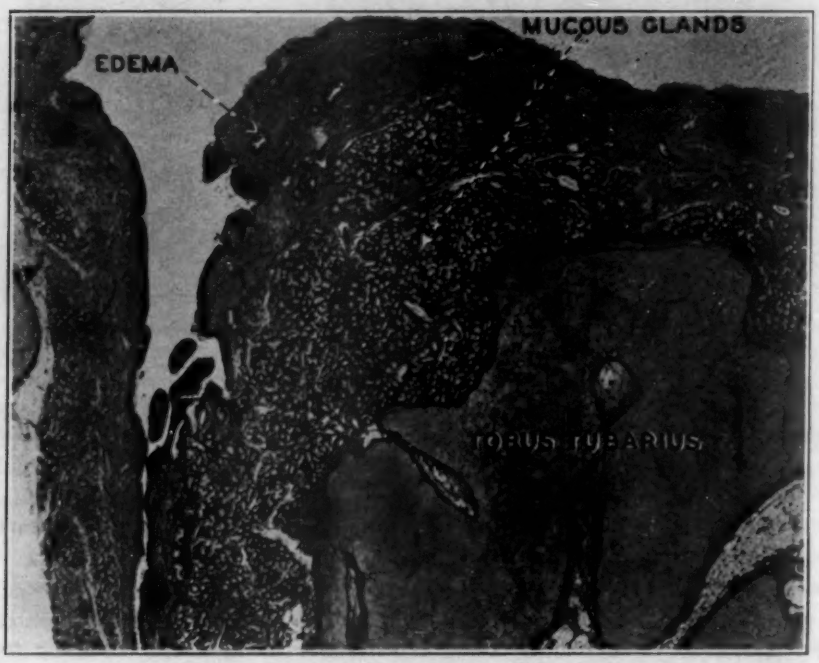


Fig. 4.—Acute catarrhal inflammation. Hypersecretory activity of the mucous glands and goblet cells is evident. There are subepithelial edema, cellular infiltration and hyperplasia of lymphoid tissue. The entire process is reversible.

edema and moderate infiltration of the leukocytes. In allergic states eosinophils are abundant. The mucous glands respond with their maximal secretory power. The edema and the increased secretory activity of these glands produce mechanical obstruction of the eustachian tube. The entire inflammatory process is one which will resolve with the minimal proliferation of fibroblasts, the eustachian tube returning to its normal patency once the irritating factor has been removed.

Acute Purulent Inflammation.—Acute purulent inflammation (fig. 5) is characterized by the infiltration of polymorphonuclear leukocytes without liquefaction of tissue. The irritation produced by the bacterial toxins is more severe than it is in acute catarrhal inflammation. There are, therefore, greater inflammatory response and greater stimulation to the proliferation of fibroblasts. If the inflammatory response is moderate, the entire process may resolve with little resultant change in the subepithelial tissue or the patency of the eustachian tube. If, however,

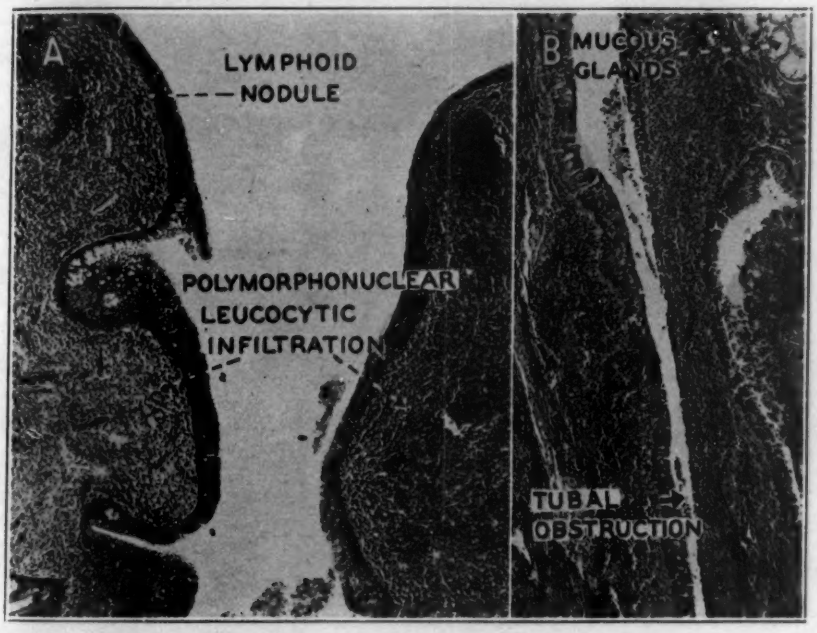


Fig. 5.—The infiltration of leukocytes is the most characteristic feature of acute purulent inflammation. There is no liquefaction of tissue. The mucous glands are hypersecretory, the lymphoid nodules are hyperplastic. The lumen of the eustachian tube is occluded by the inflammatory edema. Severe and repeated infections are followed by cicatricial narrowing of the lumen of the tube.

the inflammatory process is severe or there are repeated infections, the proliferation of fibroblasts will be great, with cicatricial narrowing of the lumen of the eustachian tube.

Acute Suppurative Inflammation.—Acute suppurative inflammation is characterized by the liquefaction of tissue in addition to the acute inflammatory response. This severe necrotizing process is most frequently seen in cases of otitis media complicating scarlet fever. Microscopic examination (fig. 6) reveals destruction of the epithelial surface with liquefaction, necrosis and heavy polymorphonuclear leukocytes, infiltration of the subepithelial tissue with evidence of local necrosis and beginning abscess formation. The abscess area must necessarily heal by the ingrowth of new blood vessels and fibroblasts. These fibroblasts at first lie about rather loosely in the subepithelial tissue. Later fibrils develop in their cytoplasm,

and the cells arrange themselves in roughly parallel form and produce an annular band of scar tissue, which eventually contracts and encroaches on the lumen of the eustachian tube.

The proliferation of fibroblasts and the production of contracting bands of scar tissue may be compared to the cicatricial obstruction to the lumen of the vermiform appendix. In the appendix the occlusion of the lumen of the tube results in inadequate drainage from the distal portion of that tube and in obstructive appendicitis. In the eustachian tube such cicatricial contractions are responsible for the relative isolation of the distal portion of the tube and the middle ear from the nasopharynx. The mucus from the middle ear and the distal isolated segment of the eustachian tube must then flow outward into the external auditory canal, with production of what is known clinically as a "tubal ear."

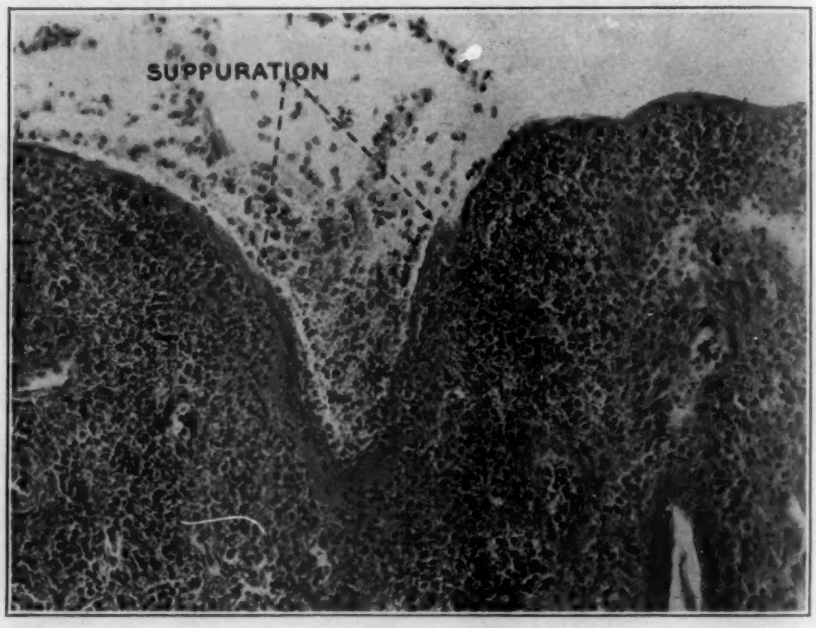


Fig. 6.—Acute suppurative inflammation is characterized by tissue necrosis and liquefaction. The necrotic area must heal by scar tissue, which encroaches on the lumen of the tube and impairs the ventilation of the middle ear.

Chronic Inflammation.—Chronic inflammation of the eustachian tube is the most frequent cause of persistent disease of the middle ear. This chronic inflammation may be the result of any inflammatory process within the nose or nasopharynx which has become chronic. Deviation of the nasal septum, chronic infection of the paranasal sinuses, all forms of allergic rhinitis and antagonistic environmental air are the most frequent causes of chronic infection of the nose and nasopharynx. Chronic inflammation differs from an acute inflammatory process in the fact that there are minimal vascular and cellular response and maximal proliferative response. Boyd [4] stated that the most significant feature of chronic inflammation is the proliferation of fibroblasts and a gradually increasing production of fibrous connective tissue. Such increased production of fibrous connective tissue in any tubular struc-

4. Boyd, W.: Textbook of Pathology, Philadelphia, Lea & Febiger, 1938, pp. 95-142.

ture, whether it is in the appendix, the urethra or the eustachian tube, will by its contraction eventually result in the narrowing of the lumen of the tube. If this narrowing develops within the eustachian tube, it is manifested by chronic disease of the middle ear. It is by this proliferation of fibroblasts that a low grade chronic inflammatory process in the eustachian tube throughout life will eventually result in cicatricial narrowing of the lumen of the eustachian tube, producing the dry chronic tubotympanitis so frequent in elderly persons (fig. *7B*). Chronic inflammation in the nasopharynx and eustachian tube stimulates the formation and hyperplasia of subepithelial lymphoid nodules. This lymphoid response to an inflammatory process is most pronounced during childhood and early adult life, although it may be present at any age. The greatest response of the lymphoid tissue takes place in the adenoid tissue on the posterior superior wall of the nasopharynx. However, in all cases there is some response of the lymphoid tissue on the lateral wall of the nasopharynx.

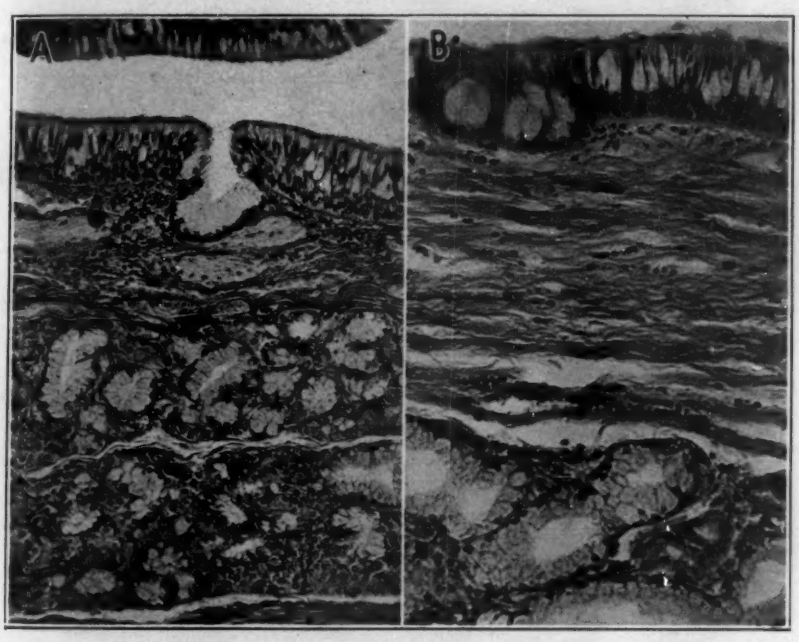


Fig. 7.—Chronic catarrhal inflammation. Proliferation of fibroblasts is one of the most prominent characteristics of chronic inflammation. The edema, hypersecretory activity of the glands, cellular infiltration and lymphoid hyperplasia respond to treatment. However, the proliferation of fibroblasts persists as subepithelial scar tissue, encroaching on the lumen. *A* demonstrates moderate proliferation of fibroblasts, while in *B* the inflammatory process has nearly subsided, leaving the organized fibrous connective tissue in the subepithelial layer.

When the adenoid tissue has been removed, there is even greater response of the lymphoid tissue on the lateral wall of the nasopharynx, in the fossa of Rosenmüller and in and about the pharyngeal orifice of the eustachian tube. In a chronic inflammatory process this hyperplastic lymphoid tissue offers additional mechanical obstruction to the eustachian tube.

Microscopically (fig. *7*) chronic inflammation of the eustachian tube is characterized by increased secretory activity on the part of the goblet cells and the subepithelial mucous glands. There are proliferation of the subepithelial connective tissue and a change in the organization of this tissue from loose areolar to dense

white fibrous connective tissue. There are subepithelial round cell infiltration and hyperplasia of the subepithelial lymphoid nodules. The inflammatory process thereby reduces the patency of the eustachian tube. The process is reversible, with restoration of normal patency, if proper therapy is begun before there is cicatricial narrowing of the eustachian tube. Conversely, once the scar tissue has formed the damage is irreparable.

Trauma.—Physical trauma, officiously produced, often plays an important role in inflammation and repair in the pharyngeal orifice of the eustachian tube. Trauma

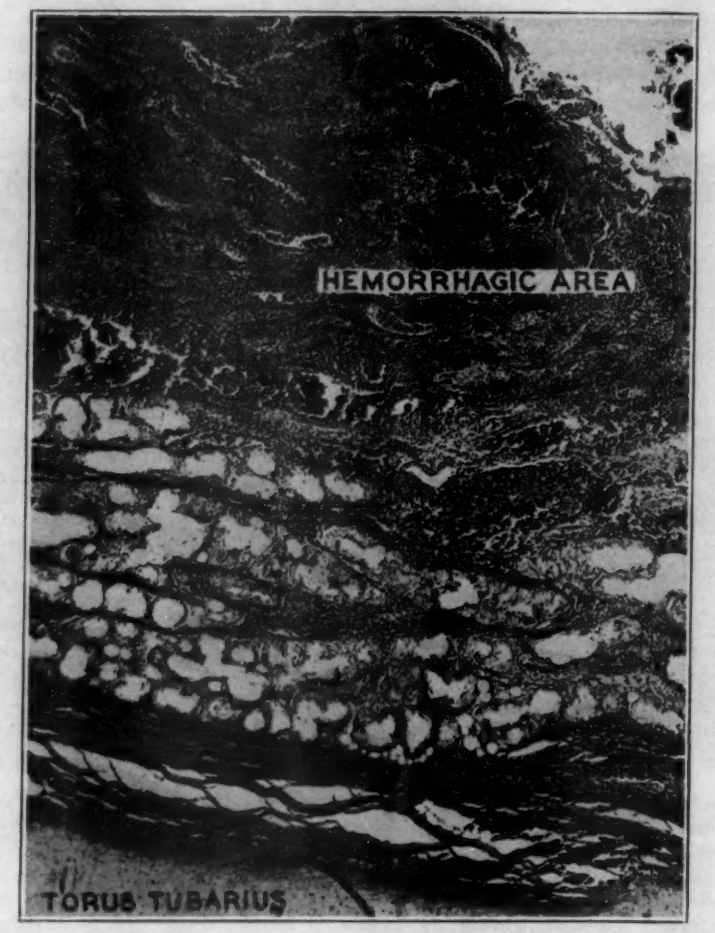


Fig. 8.—Severe subepithelial hemorrhage and inflammatory response produced by massage of the torus tubarius after adenoidectomy. The specimen was removed post mortem from a boy aged 4 years, eight hours after adenoidectomy. The process can heal only by extensive scarring. A comparable hemorrhagic area must necessarily result from manual dilatation of the eustachian tube.

of the eustachian tube may be produced by catheterization or by manual dilation of or damage to the pharyngeal orifice of the eustachian tube during the careless performance of an adenoidectomy. Trauma to the eustachian tube (fig. 8), such as is produced by manual dilation or rough instrumentation, can result only in destruction of the epithelial surface, dilatation of the subepithelial vessels and subsequent edema, cellular infiltration and fibrin formation in and on the subepithelial

tissues. If the trauma is sufficiently severe there will be hemorrhage into the subepithelial tissues. The traumatized area is subject to infection. There are associated necrosis and suppuration. The entire process must necessarily heal by the ingrowth of new blood vessels and fibroblasts, with the eventual formation of contracting scar tissue. Cholesterol crystals left from the extravasation of blood remain in the subepithelial tissues as foreign bodies, stimulating the reaction of foreign body giant cells. The extent of the damage produced by the trauma will be dependent on the severity of the physical irritant and the status of the tissues at the time the trauma is inflicted. Minimal trauma during an acute inflammatory process will produce a maximal response, while relatively severe trauma during a quiescent period will produce a minimal response. Thus, the mere catheterization of the eustachian tube (fig. 9) may result in ulceration of the epithelium, permitting

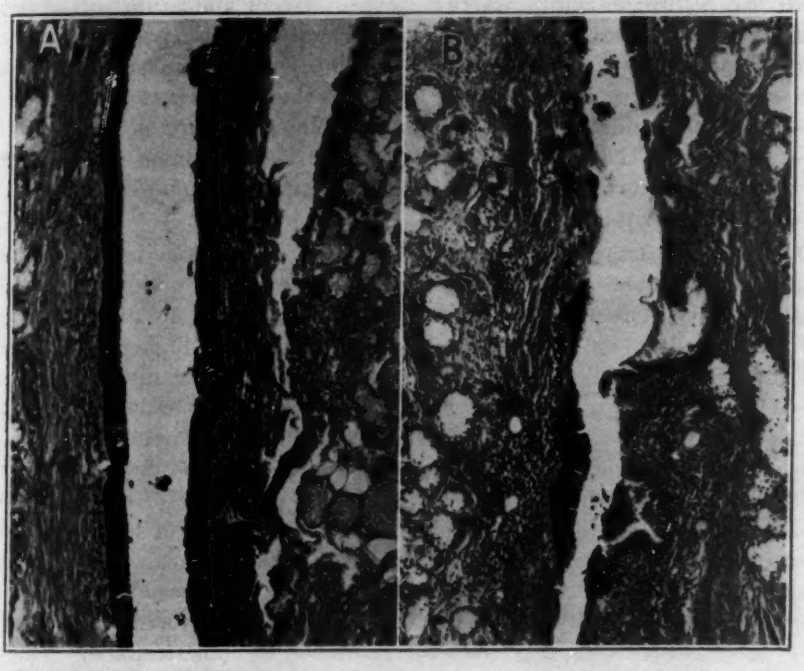


Fig. 9.—Traumatic effect of cathetherization. The patient died twenty-four hours after catheterization of the left eustachian tube for tubotympanitis. *A* reveals the intact epithelium of the right eustachian tube; *B*, loss of epithelium and the inflammatory response in the left eustachian tube.

the entrance of micro-organisms into the subepithelial tissues, and a maximal response in the subepithelial tissues. The mild catarrhal inflammatory process is thereby converted into a destructive suppurative process, which can heal only by cicatrization. It is reasonable to assume that scar tissue which results from catheterization of the eustachian tube, manual dilation of the eustachian tube and other forms of officious trauma is responsible for many cases of irremediable cicatricial narrowing of the eustachian tube.

The end result of an inflammatory process may be repair largely by resolution or by proliferation. If repair takes place mainly by resolution, the inflammatory exudate is absorbed, there is minimal proliferation of fibrous connective tissue and the normal patency and normal function of the eustachian tube are restored. If repair takes place mainly by proliferation, there result cicatricial narrowing of the

lumen of the eustachian tube and inadequate ventilation of the middle ear. Acute catarrhal and minimal acute purulent inflammation are reversible, healing being by resolution with no sequelae. Severe and repeated acute purulent inflammation, primary acute suppurative inflammation, acute suppurative inflammation induced by trauma during an acute infection, subepithelial hemorrhage produced by trauma and chronic catarrhal inflammation heal by proliferation and are irreversible, cicatricial narrowing of the eustachian tube remaining as a sequela.

CLINICAL MANIFESTATIONS

Inflammation within the eustachian tube is manifested by disease of the middle ear. This disturbance in the middle ear may vary from slight retraction of the tympanic membrane to profound deafness or chronic suppurative otitis media. The clinical picture in acute catarrhal, acute purulent and acute suppurative otitis media needs no discussion. It is the recurrent and chronic manifestations of tubal obstruction which demand particular description. Although mechanical obstruction to the eustachian tube is the primary cause of these recurrent and chronic forms of disease of the middle ear, the presenting clinical picture is dependent on the dominant factor in the histopathologic picture. This dominant factor may be the mechanical obstruction to the eustachian tube or the superimposed catarrhal inflammation or destructive infectious process.

Mechanical obstruction to the eustachian tube is first manifest by retraction of Shrapnell's membrane and a sensation of stuffiness in the middle ear. Crowe and associates [5] have demonstrated that these early manifestations of tubal obstruction are accompanied by an audiometric hearing loss for the higher tone frequencies. If the underlying pathologic condition is not corrected, the disease of the middle ear will progress to gross retraction of the tympanic membrane and symptomatic conductive deafness. Continuation of the mechanical obstruction may result in a dry perforation in the tympanic membrane, most frequently in Shrapnell's membrane, permitting entrance into the epitympanum. The end stage of this dry, prolonged mechanical obstruction to the eustachian tube is the ingrowth of squamous epithelium through the dry attic perforation, producing a cholesteatoma. Dependent on the duration and degree of the mechanical obstruction to the eustachian tube, the disease of the middle ear may stop at any stage of the aforedescribed process or may progress to the end stage with the formation of cholesteatoma. The primary mechanical obstruction may be the result of hypertrophied adenoid tissue in childhood, of chronic catarrhal inflammation with superimposed lymphoid hyperplasia, of chronic inflammation alone or of cicatricial stenosis of the eustachian tube.

When the catarrhal manifestations of the pathologic condition of the eustachian tube are such that there is an overflow of mucus into the middle ear, the clinical picture is altered. The tympanic membrane is dull and full, evidence of fluid within the middle ear. The patient complains of stuffiness and crackling noises within the ear. During a quiescent period the appearance of the tympanic membrane may return almost to normal. However, with every acute infection of the upper respiratory tract there is an exacerbation of the disease of the middle ear. Recurrent attacks of otitis media are frequent. Loss of hearing is variable. If the mechanical obstruction to the eustachian tube and the catarrhal inflammation are sufficiently severe, a perforation develops in the tympanic membrane, and there is chronic mucoid discharge from the middle ear. This type of chronic otitis media may

5. Crowe, S. J., and Baylor, J. W.: The Prevention of Deafness, J. A. M. A. **112**: 585-590 (Feb. 18) 1939.

become dry with local therapy. However, the discharge will almost invariably recur with each acute infection of the upper respiratory tract.

Aero-otitis-media, with the associated conductive deafness, is a clinical manifestation of inadequate ventilation of the cavity of the middle ear. Aero-otitis-media may develop in the presence of a normal eustachian tube when a rapid descent is made without swallowing. Aero-otitis is, however, more likely to develop in the presence of any form of inflammatory process within the eustachian tube or when there is cicatricial narrowing of the eustachian tube, the result of some previous inflammatory process or trauma.

Whenever suppuration of the middle ear is superimposed on a pathologic condition of the eustachian tube, chronic disease of the middle ear and mastoid is likely to develop, in time necessitating a radical mastoidectomy. After the radical operation has been performed, if the mechanical obstruction of the eustachian tube persists, that portion of the eustachian tube lateral to the mechanical obstruction will continue to discharge mucus into the radical mastoidectomy cavity. Proper therapy, preferably before operation, of the nose and the pharyngeal orifice of the eustachian tube is the only method which will tend to reduce the incidence of the moist radical mastoidectomy cavity.

THERAPY

The purpose of therapy of the eustachian tube is to restore the normal patency without producing irreparable damage. All forms of therapy must then be directed toward promoting the resolution of the inflammatory process. Mechanical aids with the purpose of symptomatic relief are to be employed only when there is no element of trauma.

Acute catarrhal inflammation of the eustachian tube accompanying an acute infection of the upper respiratory tract or an attack of allergic rhinitis should be treated with absolute conservatism. The treatment is that of an acute disease of the upper respiratory tract plus any local measures which will promote resolution of the edema of the eustachian tube. Hot saline gargles may be used to promote hyperemia and resolution. Chewing of gum and repeated yawning will tend to open the eustachian tube. More important than the active therapy of acute catarrhal inflammation is the avoidance of any trauma to the eustachian tube, for catheterization of the eustachian tube during the time of an acute catarrhal inflammatory condition may induce a suppurative process within the eustachian tube, resulting in the formation of obstructing scar tissue.

Acute purulent and acute suppurative inflammation of the eustachian tube have, fortunately, never been treated by traumatizing local measures. Chemotherapy for acute suppurative otitis media and related infections of the upper respiratory tract must necessarily have a beneficial effect on the eustachian tube. The soft tissue of the eustachian tube in which the inflammatory reaction occurs is sufficiently supplied with blood vessels to be affected by the chemotherapeutic agent, the local effect of which is to correct the mechanical obstruction to the eustachian tube by removal of the inflammatory edema. Once the mechanically obstructing inflammatory edema has been eliminated, adequate drainage is established, which facilitates the resolution of the infectious process within the middle ear. The second, and most important, effect of chemotherapy on the eustachian tube is reduction in the incidence of sequelae through prevention of destruction of the epithelium and the subepithelial tissue by a suppurative process which can heal only by formation of fibrous connective tissue, with its resultant partial cicatricial stenosis of the tube. It is this effect of chemotherapy which holds promise in reducing the incidence of the lifelong sequelae of necrotizing infection of the middle ear and eustachian tube.

Chronic catarrhal inflammatory processes of the eustachian tube and the resultant lymphoid hyperplasia present the most difficult therapeutic problem. The rational approach to this problem is, first, removal of the cause of the chronic inflammatory process and, second, promotion of the resolution of the inflammatory process within the eustachian tube. To remove the cause of chronic inflammation of the eustachian tube attention must be directed toward any existing pathologic condition in the patient's nose or paranasal sinuses. An all-out effort should be made to correct any existing disturbance in the upper respiratory tract, any allergic phenomena and any antagonistic factor in the patient's environmental air. As Crowe has pointed out, small doses of radiation are a definite adjunct in the treatment of partial occlusion of the eustachian tube.[6] Irradiation affects both the inflammatory process and the lymphoid hyperplasia. The 30 to 50 milligram hours of radiation used is not enough to inhibit directly the activity of the mucous glands. Small doses of radiation result in disintegration of the leukocytes and in improved circulation. By this means absorption of the cellular exudate is facilitated and resolution of the inflammatory process speeded. The hypersecretory activity of the mucous glands subsides with the inflammatory process. These small doses of radiation do not produce necrosis of normal tissue, which would call forth the proliferation of fibroblasts. The lymphatic tissue is extremely susceptible to small doses of radiation. Histopathologically, there are degeneration of the lymphoblasts and lymphocytes within the lymph follicles and absorption of the product of this cellular degeneration. Reduction in the size of the lymphoid tissue results. Clinically, this rapid shrinking of lymphoid tissue is more frequently observed through the roentgenographic demonstration of the involution of the thymus after small doses of radiation. Within the eustachian tube the reduction in the size of the lymphoid nodules and the resolution of the inflammatory process restore the normal patency and physiologic activity of the eustachian tube.

Contrary to the therapeutic effect of irradiation, repeated catheterization of the eustachian tube for a chronic inflammatory process will, by the repeated, though minimal, trauma, promote the proliferation of fibroblasts and the cicatricial narrowing of the lumen of the eustachian tube. The temporary symptomatic relief which can be produced by catheterization does not warrant this trauma to the eustachian tube during an active inflammatory process. Removal of the cause of the inflammatory process and small doses of radiation constitute the conservative, nontraumatic regimen for the treatment of chronic catarrhal inflammation of the eustachian tube. If the exciting pathologic process is an active inflammatory one, proper therapy may restore normal patency of the eustachian tube. If, however, the inflammation of the eustachian tube has progressed to cicatricial narrowing of the eustachian tube, no form of therapy can restore the normal patency. Treatment should be directed first to any pathologic condition in the nose and paranasal sinuses and, second, to the pharyngeal orifice of the eustachian tube. Irradiation in small doses is, again, a satisfactory method of restoring the normal patency of the eustachian tube. A course of inflations or any form of local trauma which produces scarring of the eustachian tube should be avoided. With flyers, particularly, all traumatizing therapy should be avoided, for the resultant cicatricial narrowing of the eustachian tube may interrupt their career.

---

6. The dose of radiation applied to the pharyngeal orifice of the eustachian tube may vary from 30 to 50 milligram hours. This dose may be delivered by radium, radon or roentgen rays. The technic of the application is discussed in the articles by Farrior and Richardson (The Nasopharyngeal Radium Applicator, Arch. Otolaryng. **35**:811-812 [May] 1942) and by Crowe and associates.

Cicatricial stenosis of the eustachian tube, the end result of an inflammatory process in or trauma to the eustachian tube, can be treated only by mechanical means. In cases of such a lesion all that can be expected of therapy is temporary symptomatic relief. Catheterization and bougienage give such relief. As the histopathologic process in cicatricial stenosis is irreversible, the symptomatic relief justifies the use of this traumatizing mechanical measure.

CONCLUSION

In conclusion, the purpose of all treatment to the eustachian tube is restoration of its normal patency. This purpose is achieved by removing the cause of the inflammation and increasing the resolution of the inflammatory process. In cases of acute catarrhal inflammation treatment of the acute infection of the upper respiratory tract and local heat constitute an effective nontraumatic regimen. In the treatment of acute purulent and acute suppurative inflammation of the eustachian tube, chemotherapy reduces the inflammatory edema and inhibits the progression of the process toward necrotizing suppuration. Chronic catarrhal inflammation of the eustachian tube should be treated by correction of any pathologic condition in the nose or paranasal sinuses and by small doses of radiation. Catheterization of the eustachian tube should not be used as a therapeutic measure in the presence of any active inflammatory process, as the resultant trauma promotes the formation of scar tissue. Catheterization of the eustachian tube should be used only as a diagnostic procedure or in the treatment of cicatricial narrowing of the eustachian tube when the histopathologic changes are irreversible.

SUMMARY

1. The histopathology of the eustachian tube is presented, with consideration of the relationship to therapeutic problems.

2. The effect of an inflammatory process on the epithelium and stroma of the eustachian tube is presented.

3. The microscopic pathologic picture is presented for acute catarrhal inflammation, acute purulent inflammation, acute suppurative inflammation, chronic catarrhal inflammation and the inflammatory process produced by trauma.

4. The lymphoid response to inflammation is discussed.

5. Therapeutic problems related to the various inflammatory reactions are discussed.

6. The effects of chemotherapy on the eustachian tube are discussed.

7. The effects of irradiation on inflammatory and lymphoid tissue are presented.

8. The histopathologic changes in the eustachian tube in aero-otitis-media and the therapy are presented.

9. Cicatricial stenosis of the eustachian tube is presented as the end result of inflammation or trauma.

10. From the histopathologic point of view, it is concluded that a nontraumatizing, conservative therapy is the rational regimen for treatment of disease of the eustachian tube.

298. General Hospital, United States Army, A.P.O. 508, % Postmaster, New York.

PREOPERATIVE DETECTION OF A BLEEDING TENDENCY IN PATIENTS WITH OTOLARYNGOLOGIC DISORDERS

CAPTAIN GILBERT N. HAFFLY (MC), U.S.N.

The results of hundreds of "routine" determinations of the blood-clotting time prior to tonsillectomy obtained by several investigators [1] have led to the joint conclusion that no coagulation test can act as an efficient diagnostic aid in the avoidance of postoperative hemorrhage. Numerous comments in the literature regarding the fallacies concerned with measurements of coagulation time have made operators skeptical of the value of preoperative tests on patients suspected of harboring a hemorrhagic tendency.

This conclusion has led to a division of opinion, particularly among otolaryngologists, with regard to the use and interpretation of the coagulation test. A large group of operators place no confidence in the test and yet employ it not only as a means of reassuring the informed type of patient but in the mistaken notion that such a test is necessary as a refuge from any medicolegal problem that might arise from fatal postoperative hemorrhage. During the last twenty years [2] no malpractice suits against physicians have been published in *The Journal of the American Medical Association* in which it has been alleged that the death of the patient was due to omission of a preoperative coagulation test.

A smaller number of operators place little confidence in the test and use it, reservedly, only when the family or the personal history, the physical signs or all of them are suggestive of a bleeding diathesis. There remains a limited number who place considerable reliance on the coagulation test, when properly performed, and who use it with discrimination and forethought.

The literature of a half-century ago is replete with ingenious methods of measurement of the coagulation time of the blood. Constant discrepancies in results obtained by the various technics employed led to the discovery of a "new method" by every able scientist interesting himself in the dilemma. Each method was designed to reduce or eliminate undesirable "extrinsic" physical factors, considered the greatest contributing elements in the variability of the coagulation time. It remained for Howell,[3] in 1912, to point out that the greatest single factor lay in the source of the blood. Prior to his revelation that venous blood obtained atraumatically by venepuncture and without the admixture of tissue juices furnished the only reliable source of blood for the test, scientists had lost themselves in the maze of error that attended the use of capillary blood obtained by simple cutaneous puncture. A year later (1913) Lee and White [4] offered a practical adaptation of Howell's method. In their article they cited a case of hemophilia in which tests "gave a coagulation time of 50 minutes by the arm-vein method, but blood from the ear clotted in 5 minutes."

From the Department of Otolaryngology, Northwestern University Medical School.

1. McKinney, R.: Coagulation Test, Arch. Otolaryng. **6**:158-161 (Aug.) 1927. Kleinert, M. N.: Coagulation Time Before Tonsillectomy, Ann. Otol., Rhin. & Laryng. **37**:977-980 (Sept.) 1928.

2. Holloway, J. W.: Personal communication to the author.

3. Howell, W. H.: Nature and Substance of the Thromboplastic Substance of the Tissues, Am. J. Physiol. **31**:1-21, 1912.

4. Lee, R. I., and White, P. D.: A Clinical Study of the Coagulation Time of the Blood, Am. J. M. Sc. **165**:495-503, 1913.

After Lee and White's enumeration of the many advantages accruing to venepuncture as compared with cutaneous puncture, one might have expected a wealth of substantiating clinical evidence to flood the literature in its wake. Instead, most investigators ignored the work of these authors and continued to err in their faulty conclusions, based, as before, on the unscientific use of capillary blood. As recently as 1927 Hunt [5] reported on the coagulation methods required prior to tonsillectomy by 200 recognized hospitals in the United States. Twenty-two methods were listed, of which a generous estimate of less than 10 per cent required the use of venous blood.

MATERIAL AND METHODS

Three proved cases of hemophilia encountered in the otolaryngologic, ophthalmologic and oral surgical wards of Cook County Hospital are presented, with particular emphasis on the disparity existing between the coagulation time obtained on capillary blood and that on venous blood with the use of similar methods. In each case the platelet count, the clot retraction and the prothrombin time were normal. The response to the capillary resistance test of Rumpel and Leede was normal in each instance. Duke's procedure was employed for determination of the bleeding time.

Brief mention should be made of the methods used in determination of the coagulation time of each type of blood. A single method, the Wright capillary pipet technic, was used for capillary blood. Two methods, the Wright capillary pipet technic and the Lee and White method, were used for venous blood. A brief description of the methods used for determination of the coagulation time follows.

Capillary Blood.—Wright Capillary Pipet Method: The lobe of the ear is pierced deeply with a clean, sharpened cutting needle so that the released blood falls in free droplets. Six capillary tubes, calibrated to receive a column of blood of 5 cm. with a diameter of 0.25 mm., or 0.01 inch, are filled in quick succession, and the time of filling is noted. These filled tubes are then laid flat in order on a piece of gauze. Beginning with tube 1, the pipet is carefully broken at thirty second intervals on filter paper in lengths of 1 cm., and the presence or absence of a clot in the form of a shred of fibrin is carefully noted. The normal coagulation time as determined by this method lies between two and a half and five minutes.

Venous Blood.—Wright Capillary Pipet Method: The procedure is the same as that given for capillary blood.

Lee and White Method: Three small test tubes (Widal type) about 10 mm. in diameter are cleaned first with cleaning solution and rinsed thoroughly with tap water and then with an isotonic (0.85 per cent) solution of sodium chloride. Venous blood is obtained atraumatically (without admixture of tissue juices) in the usual manner, the blood being allowed to enter the syringe without suction and care being taken to avoid air bubbles. About 1 cc. of blood is transferred to each tube. At the end of every thirty seconds the first tube is gently inclined until it is possible to invert the tube without any flow of blood. The second tube is then inclined every thirty seconds until the blood in that tube has coagulated. The process is then repeated with the third tube. The longest interval between the first appearance of blood in the syringe and the definite coagulation of blood in one of the tubes is taken as the coagulation time. The coagulation time of the blood of normal adults as determined by this method varies from about five to ten minutes.

RESULTS

Brief summaries of the history and the pertinent physical observations in the 3 cases of hemophilia are now reported.

CASE 1.—F. P., aged 20 years, who was known to be hemophilic, entered Cook County Hospital on Feb. 4, 1941, suffering from spontaneous hemorrhage into the tongue and the floor of the mouth, including the submaxillary and submental regions. The family history was not remarkable except that an older brother had been troubled for years with "rheu-

5. Hunt, W. M.: Coagulation Time Prior to Tonsillectomy, Ann. Otol., Rhin. & Laryng. **36**:187-200 (March) 1927.

matism." The patient's past history included numerous severe attacks of spontaneous nasal hemorrhage and frequent "rheumatic attacks" in the shoulder joints. Other pertinent physical observations included partial ankylosis of the right wrist with a moderate "forklike" deformity.

CASE 2.—J. P., aged 22 years, a brother of F. P. (case 1), was not aware that he was hemophilic, but readily admitted having had "rheumatic attacks" in all his joints except those of the fingers since childhood. Because he bruised easily, with painful swelling of his knee joints, even on moderate exercise, he had never indulged in vigorous sports. Physical observations included partial ankylosis of the right elbow and the left knee joint. Scars in both cubital fossae suggested strongly that blood transfusions had been performed in early childhood. The patient recalled that he had been hospitalized at an early age for a serious illness, but he knew none of the specific details. There were old hemarthroses of the wrists and a large recent bruise on the left forearm.

CASE 3.—H. L., aged 24 years, was admitted to the ophthalmologic ward of Cook County Hospital on March 16, 1941 with a history of enucleation of the right eye, which had been blind and painful, at a local private hospital three weeks before. There had been an intermittent, but almost constant, slow hemorrhage from the socket since the time of operation. The patient and his relatives were insistent that the surgeon had been warned that the patient was known to be a "bleeder" but that he had reassured them after making a "blood test," performed with "tiny glass tubes" on a "drop of blood obtained by sticking the right ear." Several attempts, both surgical and medical, to stop the hemorrhage had been unsuccessful prior to the patient's entrance to Cook County Hospital. Aside from an old swelling of the right knee joint, which he had suffered while diving, there were no other pertinent physical signs. The patient stated that the only exercise he indulged in was swimming, as he tended to "bruise easily."

The following tabulation of data in these 3 cases is self explanatory.

			Coagulation Time		
			Capillary Blood,	Venous Blood	
Case No.	Initials	Duke Bleeding Time, Min.	Wright Method, Min.	Wright Method, Min.	Lee and White Method, Min.
1	F. P.	7*	7	50.5	97.5
2	J. P.	3.5	4	28.5	55.5
3	H. L.	4	4.5	27.5	42.5

*Delayed bleeding from the pricked ear followed in four hours and persisted seven hours. It was controlled with difficulty by collodion and cotton dressings plus a pressure clamp.

The tabulation shows at a glance that a marked discrepancy exists between the coagulation time of capillary and that of venous blood when they are measured by a similar method (Wright capillary pipet method). The delicacy of the Lee and White method is indicated by the difference in results obtained with it and with the Wright capillary pipet method on venous blood.

COMMENT

Quick [6] has pointed out that although a significantly retarded coagulation time clearly indicates that "something is amiss," it offers no clue to the factor at fault. There may be a deficiency in prothrombin, fibrinogen or platelets, or, theoretically, an undue increase in antithrombin or some heparin-like factor. It has also been pointed out that a normal coagulation time, on the other hand, offers no assurance that the coagulation time is in fact normal. It is contended that the normal fibrinogen, thromboplastin or prothrombin content of the blood is enormously in excess of that actually required for coagulation and that a significant diminution in any one of these factors may be associated with a coagulation time which falls within normal limits.

6. Quick, A. J.: Classification of Hemorrhagic Diseases Due to Defects in the Coagulation Mechanism of the Blood, Am. J. M. Sc. **199**:118-132 (Jan.) 1940.

However, since the determination of the coagulation time, properly performed, can be demonstrated to be of definite diagnostic value, particularly in the detection of unrecognized hemophilia, it is apparent that it should either be meticulously carried out on venous blood or be permanently discarded as a worthless and misleading diagnostic procedure when employed with capillary blood. Osgood's [7] comment is typical of the hematologist's view:

> The coagulation time is usually determined as a routine procedure before any operation, and in all cases in which a hemorrhagic disease is suspected. It is practically of no value unless determined by the Lee and White method on blood obtained from the vein, since even hemophiliacs may have normal clotting times if other methods are used.

To assume immediately that a prolongation of the coagulation time is evidence of hemophilia is to labor under a misapprehension. Kugelmass [8] had this to say:

> Hemophilia, a clinical chimera for many a bleeding problem, is too rare a condition to cover so many hemorrhagic disturbances, is too hereditary a disease to include so many mature bleeders, is too well defined a disease entity to become a haphazard label of hemorrhagic confusion. A solitary hemorrhage for the first time requires that every local cause be excluded before a diagnosis of hemophilia is ventured. Profuse hemorrhage at birth is varied in origin unless inheritance makes hemophilia a consideration. A prolonged clotting time alone is never pathognomonic of hemophilia. Such common correlations with hemophilia have confused the literature to such an extent that Bulloch and Fildes of the London Eugenic Laboratory have been able to authenticate only 44 hemophilic families of 273 reported in a century of literature.

Goldstein [9] stated that true hemophilia is a sharply differentiated clinical entity, characterized by normal bleeding time, normal prothrombin time, restricted occurrence in males, hereditary transmission according to the so-called Lossen law, absence of demonstrable vascular changes and greatly prolonged coagulation time. He added that "this last and basic differential symptom is no longer considered as a characteristic and as the single pathogenic factor in hemophilia."

It must be emphasized at this point that the majority of patients with a bleeding tendency do not have a prolonged coagulation time, and therefore do not have hemophilia. For this reason, any reliance on the test of the coagulation time of the blood as a routine and solitary preoperative laboratory procedure must be condemned.

It is my conviction that routine tests of the clotting mechanism of the blood are not indicated unless the family or the personal history or the physical signs are suggestive of a bleeding diathesis. In his excellent treatise on slight bleeding tendencies and latent bleeders, Roskam [10] maintained that a careful personal history furnishes the most important single diagnostic clue. He stated that, in addition to the family history, he makes pertinent inquiry into the following symptoms: (1) prolonged bleeding following small wounds or minor operations, including the extraction of teeth; (2) easy bruising; (3) epistaxis; (4) purpuric spots; (5) subconjunctival hemorrhages; (6) "rheumatic pains"; (7) melena; (8) hematuria; (9) hemoptysis; (10) subperiosteal hemorrhages, and (11) bleeding gums. He insisted that a careful physical examination for changes in the joints, such as ankyloses and hemarthroses, ecchymoses and purpuric spots, is imperative. If the

7. Osgood, E. E.: A Textbook of Laboratory Diagnosis, ed. 3, Philadelphia, The Blakiston Company, 1940, p. 270.

8. Kugelmass, I. N.: Mechanism of Hemophilia in Infancy and in Childhood, Am. J. Dis. Child. **44**:50-68 (July) 1932.

9. Goldstein, H. I.: Hemorrhagic Blood Dyscrasias and Dysplasias: Symptomatology, Diagnosis and Treatment, Acta pædiat. **22**:485 (Sept.) 1937.

10. Roskam, J.: Slight Bleeding Tendency and Latent Bleeders: Classification and Discovery, Presse méd. **47**:861-863 (May) 1939.

history or the physical signs are suggestive, he recommended a number of diagnostic blood tests, among which he included "the venous coagulation time."

What other diagnostic aids does one have at one's disposal in determining the cause of an obscure bleeding tendency, particularly if the coagulation time of venous blood is within normal limits? In addition to Lee and White's method for determination of coagulation time of venous blood, the following procedures may be carried out with comparative ease and accuracy in the average office or hospital laboratory: (1) Duke test for bleeding time; (2) clot retraction test; (3) capillary resistance test of Rumpel and Leede; (4) platelet count, and, in selected cases, (5) determination of prothrombin time.

No strenuous attempt will be made here to indicate the proper interpretation of the results of the various tests. Quick [6] offered an excellent clinical classification of hemorrhagic diseases due to defects in the coagulative mechanism of the blood, based on the coagulation theory of Morawitz. He pointed out that on the basis of this classification a satisfactory diagnosis of a hemorrhagic disease can be made. He noted that "a prolonged coagulation time by the Lee and White method" may be due to diminished prothrombin or thromboplastin or to the presence in the blood of an anticoagulant, such as heparin.

The prothrombin time is informative in selected cases, such as those of jaundice and hepatic disease. Quick stated that hemophilia can be readily diagnosed, for the prothrombin time will be found to be normal even though the clotting time may be exceedingly delayed. The bleeding time is independent of the coagulation time. The bleeding time is slightly prolonged (five to ten minutes) in cases of severe anemia. It is greatly prolonged in (1) cases in which the platelet count is excessively reduced (ten to ninety minutes); (2) cases in which the fibrinogen content of the blood is excessively reduced (ten minutes to two hours), and (3) cases in which both platelets and fibrinogen are reduced. According to Duke,[11] the bleeding time is apparently of no value in determining the tendency to bleed in cases of jaundice and hemophilia and in cases of the types of purpura haemorrhagica in which the platelet count is normal.

In cases of a bleeding tendency of obscure origin the presence of fibrinogen, serum calcium and antithrombin may be quantitatively measured. If blood ultimately clots, the presence of fibrinogen is established. Not all hematologists are agreed that the determinations of serum calcium and antithrombin are of clinical value in the detection of a hemorrhagic tendency.

There remains a certain small percentage of cases in which all the aforementioned tests give essentially normal results and yet there is a profound bleeding tendency, with alarming hemorrhage following a surgical procedure. Into this group fall those confusing, and often unrecognized, cases of hereditary thrombasthenia (hereditary pseudohemophilia). This condition is characterized by (1) a history of bleeding in the family in either sex; (2) an approximately normal coagulation time; (3) usually a prolonged bleeding time; (4) normal clot retraction; (5) a normal or slightly prolonged prothrombin time; (6) a normal platelet count, and (7) usually a normal blood count.

While the bleeding time in cases of this condition is usually prolonged, in some instances it is normal or only slightly prolonged. Two such cases [12] have been observed within the past several years in private practice. In 1 case, that of a boy 11 years of age, severe hemorrhage followed tonsillectomy. All diagnostic blood tests, including the Duke method for measurement of bleeding time,

11. Duke, W. W.: The Relation of Blood Platelets to Hemorrhagic Disease, J. A. M. A. **55**:1185 (Oct. 1) 1910.

12. Alt, H. L.: Personal communication to the author.

had given values within normal limits. However, the family history indicated that at least 3 members (1 male and 2 females) had experienced bleeding episodes after elective surgical procedures and tooth extraction. The past history revealed that the patient had bled profusely after tooth extraction on a previous occasion. In the other case, that of a man aged 33, tooth extraction on a previous occasion had required repeated blood transfusions to control hemorrhage. The family history revealed that several siblings (male and female) had had similar, but less severe, hemorrhages after elective surgical procedures and tooth extraction. The Duke bleeding time (five minutes) was only slightly prolonged (normal three minutes). The extraction of two teeth again required repeated blood transfusions to control hemorrhage.

It must be stressed that at least in this condition, hereditary thrombasthenia, the past and the family history are more important than the diagnostic blood tests. Any operative procedures, particularly those on the teeth, nose and throat, are best avoided in patients with this disease. If surgical intervention is necessary, the operator must proceed with extreme care. Every precaution must be utilized, including the preoperative typing of the patient's blood and that of his immediately available donors.

SUMMARY

Three established cases of hemophilia are presented, in each of which there existed a remarkable discrepancy in the coagulation time of capillary blood as compared with that of venous blood when similar methods were employed.

The Lee and White method of measurement of coagulation time, employing venous blood, is a delicate test of extreme value in determination of the clotting time, provided such blood has been secured without trauma and the admixture of tissue juices.

Unless the family or personal history or the physical signs are suggestive of a bleeding diathesis, routine tests of the clotting mechanism of the blood are not indicated.

If the family or personal history or the physical signs are suggestive of a bleeding diathesis, the following tests are to be considered of diagnostic value: (1) determination of venous coagulation time (preferably the Lee and White method); (2) Duke method for measurement of bleeding time; (3) clot retraction determinations; (4) capillary resistance test of Rumpel and Leede; (5) platelet count, and, in selected cases, (6) determination of prothrombin time.

The more advanced and technically difficult tests of a quantitative nature fall in the domain of the larger private and hospital laboratories. These include the quantitative determination of (1) fibrinogen, (2) serum calcium and (3) antithrombin. The clinical value of determinations of the latter two factors is questionable.

In certain cases in which a bleeding diathesis exists, the results of all diagnostic blood tests may be normal and yet the patient may suffer fatal hemorrhage after a surgical procedure. This is particularly true in the condition known as hereditary thrombasthenia (hereditary pseudohemophilia), in which the family and the past history is frequently more important than the evidence from diagnostic tests. In all cases of such disease one must proceed with extreme care despite the lack of any positive diagnostic evidence of a bleeding tendency. Every precaution must be utilized, including the preoperative typing of the blood of the patient and that of his immediately available donors.

Dr. Howard L. Alt gave helpful suggestions in this study.

4232 Bagley Avenue, Seattle.

THE DIAPHRAGM-ROD PROSTHESIS FOR THE MIDDLE EAR

A. G. POHLMAN, M.D.
LOS ANGELES

Research on hearing, whether viewed from an academic standpoint or from the point of view of advances in the treatment of auditory disabilities, cannot be said to have been conspicuously productive. Three interpretations of the function of the middle ear in relation to the conduction of air vibrations to the cochlea had been proposed before Cotugna demonstrated the labyrinth liquid in 1774. Each of these three interpretations finds active supporters even at the present time.

Obviously, the development of an effective prosthesis for the middle ear must be based on the function of the sound conduction apparatus it is designed to replace. This makes it necessary to discuss briefly the differences in opinion on the conduction of air vibrations into the cochlea.

The three interpretations of the function of the middle ear agree that the drum membrane vibrates to air sounds and disagree on how these vibrations are conducted to the cochlea. The most commonly accepted explanation assumes that vibrations of low frequency in the drum membrane are conducted through the ossicular chain to the oval window, while vibrations of high frequency pass through the air of the middle ear to enter the cochlea through the round window membrane. This explanation was proposed to account for the seemingly greater loss in sensitivity for low tones than for high tones in cases of conduction deafness. Alexander,[1] in 1931, presented a paper in which he discussed the relative importance of ossiculocochlear and aerocochlear conduction. He stated:

> In all vertebrates in which an air-containing middle ear is present and closed to the outside and in which the auditory tubes are also functionally closed, two pathways for conduction of air sounds may be distinguished: an ossiculocochlear and an aerocochlear. The pathway for ossiculocochlear conduction is by way of the drum membrane and ossicles to the oval window and through the scala vestibuli on through the helicotrema to the scala tympani. The pathway for aerocochlear conduction is from the drum membrane through the air of the tympanic cavity to the round window and directly into the scala tympani by way of the round window membrane. Under these conditions aerocochlear conduction establishes a direct route, particularly for high frequencies, which leads to nerve stimulation without loss of intensity. With defects of the middle ear after loss of the drum membrane, the ossicles or both, aerocochlear conduction may become increasingly important, particularly when the auditory tubes are closed. The most favorable condition, anatomically, at the membrana tympani secundaria, and therefore that of the greatest efficiency in aerocochlear conduction, is found after radical mastoid operation in cases in which there is excellent hearing acuity for air-conducted sound. Here, in all cases, in spite of the otherwise excellent sensitivity, sounds up to about 50 double vibrations are not heard by air conduction.

In a discussion on Alexander's paper, I [2] pointed out that he had completely disregarded all audiometric evidence bearing on the problem published both at home and abroad. Recently Nasiell [3] expressed the opinion that the results obtained with

---

This study was aided by a grant in aid from the Carnegie Corporation.

From the Department of Otolaryngology, University of Southern California School of Medicine.

1. Alexander, G.: Monatschr. f. Ohrenh. **65**:1 and 173, 1931.
2. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **40**:717, 1931.
3. Nasiell, V.: Acta oto-laryng. **26**:608, 1938.

his middle ear prosthesis confirm the interpretation of dual conduction; this problem will be discussed at the close of this paper.

Jones,[4] in 1940, asserted that air vibrations of all frequencies are effectively conducted into the cochlea through the round window. Like Alexander, he expressed the belief that the effective thrusts on the basilar membrane come from the scala tympani aspect, and he, too, disregarded the rather extensive literature on the subject. He presented a unique explanation for the function of the drum membrane and the ossicular chain.

> The physiologic effect is that with safe pressures [in air vibrations] the manubrium and the stapes move outward, supplementing the inward movement of the secondary tympanic membrane in producing an upward movement in the basilar membrane. When, however, the pressure becomes excessive and likely to damage the delicate mechanism of the internal ear, the movement is reversed and the stapes is driven into the fenestra ovalis and the upward movement in the basilar membrane is arrested.

This means that at low intensities the ossicular chain pulls on the oval window as the pressure of the air vibrations in the middle ear pushes on the round window, and that as the intensity rises the drum membrane and the ossicular chain act as a shock absorber.

While Alexander warned investigators "not to overpower the facts," and then proceeded to do so, Jones claimed that his interpretation was "supported by irrefutable experimental evidence which can easily be verified," but he presented none of this information. He, however, expressed agreement with Alexander:

> High notes should be more readily perceived than low notes, and this is characteristic of hearing in the absence of the drum.

The third interpretation of the function of the middle ear holds that the drum membrane and the ossicular chain are important to the effective conduction of all usual audiofrequencies. In 1928 I[5] presented the results of audiometric experiments, conducted in collaboration with Dr. F. W. Kranz in 1924, which supported this conception. Our evidence has been confirmed by a number of observers and is substantiated by the results reported in the present communication. Thus, in patients in whom the drum membrane and ossicles have been lost, the condition is like that of complete conduction deafness with an intact middle ear apparatus and with loss in sensitivity for all important audiofrequencies of 40 to 50 decibels.

The fact that a conduction-deafened patient does not hear low frequencies well is a real phenomenon, but if he hears the high frequencies as well as the examiner, the result may be dependent on the presence of presbycusis in the examiner. This point may be illustrated by the following contrast between the acuity of hearing in a patient with complete conduction deafness and my own. It will be seen that in the patient (fig. 1 *A*) the acuity for air-conducted sound taken with a heavily padded receiver practically coincided with the acuity for bone-conducted sound taken from the mastoid. Figure 1 *B* shows the acuity for air-conducted and for bone-conducted sound for my better ear, and it will be noted that the completely conduction-deafened person with normal internal ears heard the 4096 and 8192 frequencies as acutely as I did. In other words, the greater the degree of presbycusis in the examiner, the more he is convinced that the conduction-deafened patient hears high frequencies well.[6]

It was formerly believed that in cases of complete conduction deafness the intact, but useless, middle ear apparatus might act as a barrier to the production of air

4. Jones, H. M.: Hearing and Equilibrium, Baltimore, Williams & Wilkins Company, 1940.

5. Pohlman, A. G.: Proc. Internat. Cong. Otol., Copenhagen, 1928, p. 324.

6. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **42**:352, 1933.

vibrations in the tympanic cavity. If this were true, then perforation of the drum membrane or removal of the middle ear apparatus should improve the acuity for air-conducted sound. Cooper [7] reported favorable results through perforation of the drum membrane, and Deleau [8] practiced ossiculectomy extensively as early as 1820. Kranz and I had the opportunity of testing a number of patients who had had ossiculectomy performed, in particular a patient who was quantitatively examined shortly before and shortly after bilateral removal of the drum membrane and outer ossicles. The case of this patient was reported [5] before the International Congress of Otolaryngology in Copenhagen in 1928, and the acuity curves are reproduced in

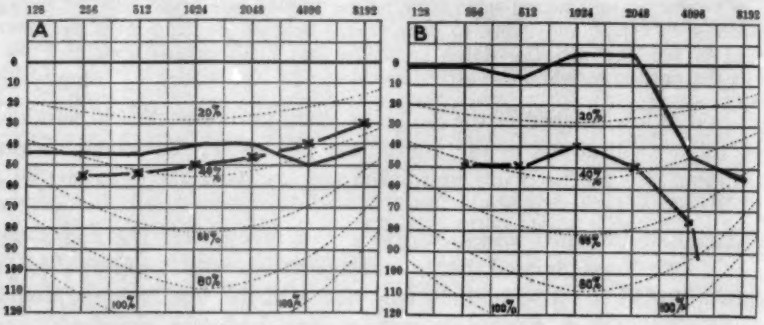


Fig. 1.—The upper line in *A* and *B* presents the acuity curve for air-conducted sound; the broken line, that for bone-conducted sound taken from the mastoid. *A* represents characteristic conduction deafness with a normal internal ear; *B*, characteristic perception deafness with a normal middle ear.

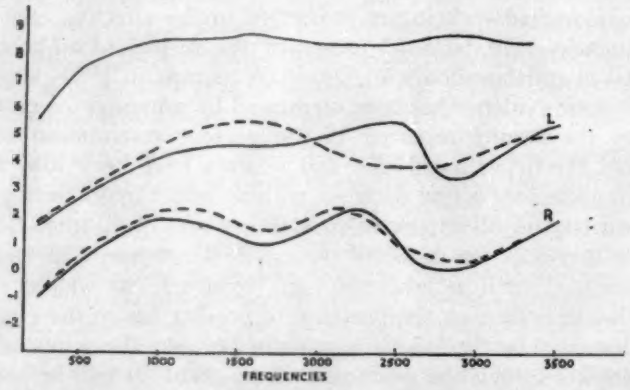


Fig. 2.—The uppermost line shows the normal sensitivity as determined by Kranz. The solid lines marked *R* and *L* were taken before bilateral ossiculectomy, and the broken line shows the effect of the operation.

figure 2. The uppermost line on this chart indicates the normal acuity for air-conducted sound as determined by Kranz [9] with a continuous frequency audiometer. The acuity curves for the right and for the left ear before ossiculectomy are shown by the solid lines *R* and *L* respectively and demonstrate practically uniform loss of

7. Cooper, A., cited by Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, F. Enke, 1913, vol. 2, p. 337.

8. Deleau, cited by Politzer, A.: Geschichte der Ohrenheilkunde, Stuttgart, F. Enke, 1913, vol. 2, p. 447.

9. Kranz, F. W.: Physiol. Rev. **3**:66, 1923.

acuity throughout the range tested. The dotted lines indicate that the ossiculectomy did not benefit the hearing on either side. This patient, however, had perforated drum membranes due to trauma and purulent otitis media. A further note on this case will be made later. The negative result of ossiculectomy in this case, however, by no means proves that no patient was benefited by the operation.

Recently a patient was observed in whom acuity for air-conducted sound was disproportionately poor as compared with that for bone-conducted sound. The drum membrane was thickened and diseased, and the suggestion was made and accepted that a perforation might increase the sensitivity to air-conducted sound. The patient was tested with a Western Electric 6 A audiometer, the air receiver being used as a loud speaker unit located about 1 foot (30 cm.) from the head. This was done to avoid the possibility of bone conduction due to vibration of the receiver at high intensities. The external auditory canal was sterilized; the drum membrane was anesthetized, and a segment of the posterior quadrant was excised by Dr. Max Pohlman. The patient immediately reported benefit, and the hearing was again tested under the same conditions as in the previous experiment. The lowest line (fig. 3) indicates the acuity before and the intermediate line the acuity after the excision. While these curves cannot be quantitatively compared with the usual acuity curves, the improvement shown is significant. A third test was made after excision with the air receiver in contact with the auricle, and the highest line shows the characteristic curve for complete conduction deafness with almost uniform loss in sensitivity for all frequencies by a factor of about 50 decibels. This case will be referred to later in relation to certain further experiments.

I [10] observed a somewhat similar result in a patient operated on by Nesfield. The Nesfield operation consists in establishing the air continuity between the outside and the tympanic cavity by creating a by-pass around the intact drum membrane. The by-pass leads from the external auditory canal into the mastoid antrum. The opening into the external auditory canal tends in time to be occluded by a membrane, and when this happens air conduction acuity is lowered. Excision of the membrane increases the acuity of hearing. This was convincingly demonstrated to Dr. Ery Luescher, of Berne, Switzerland, and me in London in 1929. Some time later one of Major Nesfield's patients was referred to me for excision of the membrane occluding the by-pass, and this made it possible for me, in collaboration with Dr. William Sauer, of St. Louis, to conduct a series of quantitative tests. The averaged increases in acuity for air-conducted sound on several occasions with the by-pass open and with it occluded are shown in figure 4, and the enhancement of air-conducted sounds was appreciable except at a frequency of 2048. This may account for the more acute hearing in some cases of mastoid fistula when the fistula is patent than when it is packed. As in the instance previously noted, this should obtain only in cases of complete conduction deafness, in which the damped drum membrane is an actual barrier to the development of air vibrations in the middle ear. These cases, however, are relatively unusual and may be illustrated by the following 2 examples. In the first case pronounced bilateral perception deafness was superimposed on complete conduction deafness on both sides. The patient claimed to hear fairly well with a bone receiver, but not with an air receiver. She stated further that she heard with an air receiver just as well when it was placed on the mastoid as when it was applied over the auricle. She was tested with a heavily padded air receiver and with a bone receiver, which tends to follow closely the curve for air conduction acuity in cases in which the Rinne reaction is negative.

10. Pohlman, A. G.: Interpretation of Conduction Deafness: Report of Two Unusual Cases, Arch. Otolaryng. **14**:48 (July) 1931.

The results are shown in figure 5, in which the curve for bone conduction acuity, particularly on the right, is at a higher level than the curve for air conduction acuity. There is little doubt that the patient would also have had material improvement in air conduction acuity by a perforation of the drum membrane. It is unfortunate, so far as experimental evidence is concerned, that this procedure could not be recommended.

A second case is that of a young man who had had a bilateral mastoidectomy. The external auditory canals were contracted and tortuous. He gave a definite

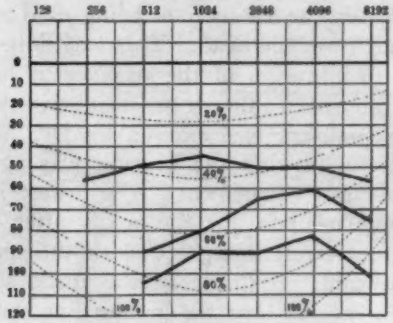


Figure 3

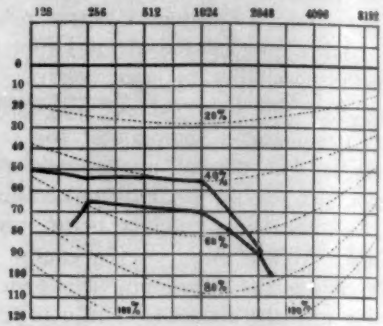


Figure 4

Fig. 3.—The lowest line indicates the sensitivity taken on the audiometer, the receiver being employed as a loud speaker unit, before excision of the drum membrane. The intermediate line indicates improvement after excision under similar conditions of test. The uppermost line presents the sensitivity when the air receiver was applied to the ear.

Fig. 4.—The lower line shows the sensitivity before excision of the diaphragm which occluded the by-pass from the external auditory canal into the mastoid antrum (Nesfield). The upper line indicates the improvement in air conduction sensitivity with the by-pass open.

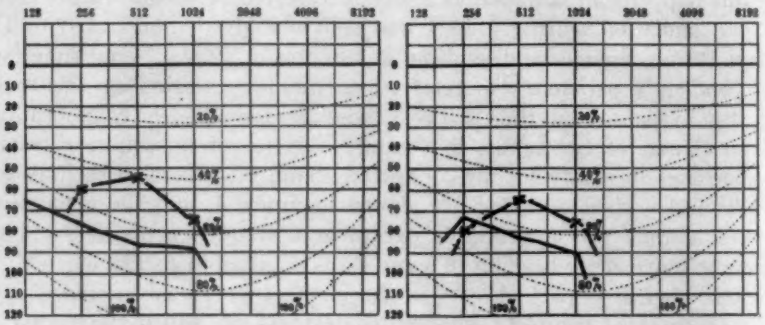


Fig. 5.—The broken line shows the sensitivity for bone-conducted sounds from the mastoid. The solid line indicates a disproportionate loss in the sensitivity to air-conducted sounds. Audiogram for the right ear is at the left; that for the left ear, at the right.

negative response to the Rinne test and a positive one to the acoustic fan. The acuity curves for air-conducted and for bone-conducted sound are shown in figure 6; as in the previous case, the air telephone was heard as acutely when placed on the mastoid as when applied to the auricle.

While complete conduction deafness ordinarily results in more or less uniform loss in sensitivity throughout the frequency range, Crowe and Baylor [11] demon-

11. Crowe, S. J., and Baylor, J. W.: Prevention of Deafness, J. A. M. A. **112**:585 (Feb. 18) 1939.

strated that the condition may produce greater disability for high tones than for low tones. The type of conduction deafness with appreciably greater losses in sensitivity for high tones than for low tones appears to be more common in children than in adults, and, seemingly, a direct quantitative correlation between air conduction and bone conduction sensitivity for extremely high tones is difficult to establish. Figure 7 shows the acuity for air-conducted sound in a patient whose one ear was normal and whose drum membrane and ossicles on the other side had been lost by reason of chronic purulent otitis media. Here, the more or less uniform loss in acuity for air-conducted sound characteristic of complete conduction deafness is evident. Figure 8 contrasts the air-conduction acuity of the two ears of a patient whose middle ear apparatus had been removed on one side and whose intact

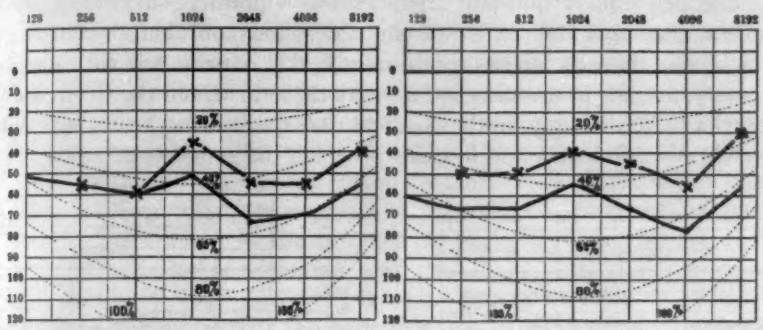


Fig. 6.—The broken line shows the sensitivity to bone-conducted sounds from the mastoid. The solid line indicates a disproportionate loss in the sensitivity to air-conducted sounds.

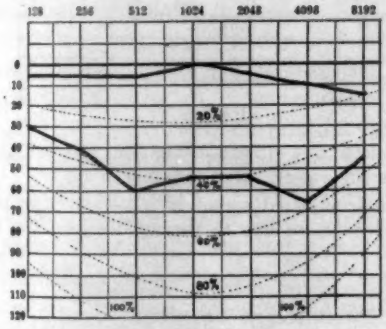


Figure 7

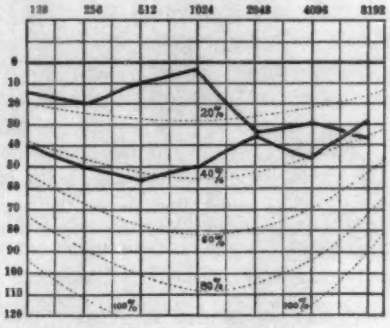


Figure 8

Fig. 7.—Sensitivity to air-conducted sound in the normal ear as compared with conduction deafness due to loss of the middle ear apparatus.

Fig. 8.—The upper line shows middle ear deafness, with relatively greater loss in sensitivity for high tones than for low tones. The lower line indicates the acuity for air-conducted sound for the other ear, on which radical operation was performed.

drum membrane showed evidences of otitis media, from which he had recovered. The acuity curve for the side of operation is the characteristic curve of complete conduction deafness, and that on the intact side, the Crowe type of conduction deafness, with relatively greater losses in sensitivity for high frequencies than for low ones. I [12] have reported a similar condition in 2 other cases. In both bone-conduction acuity as determined by the bone telephone was normal on both sides.

12. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **48**:596, 1939.

Occasionally inflation of the middle ear produces a relatively greater increase in the sensitivity to high tones than to low tones. Figure 9 shows the curves for air conduction acuity in a case of incomplete conduction deafness before and after inflation; the correction of the deformation in reception through notable improvement for high frequencies is obvious. As Crowe correctly indicated, this type of conduction deafness may be mistaken for a perception disability.

It has long been known that some patients with wide perforation of the drum membrane may receive benefit through sealing of the perforation, while others do not show an increase in acuity. Probably the latter patients already have complete conduction deafness and would not hear any more acutely than they do if the drum membrane were intact. However, when the sound conduction apparatus is functional, a large perforation not only reduces the vibratory surface of the drum membrane but decreases the thrust of the air sounds on this structure. These patients, in whom there is direct continuity of the outside air with that of the tympanic cavity, are not in the same position as those in whom the drum membrane acts as a barrier. Accordingly, in the Nesfield case the hearing acuity was enhanced when the by-pass was opened, and in cases in which the sound conduction apparatus

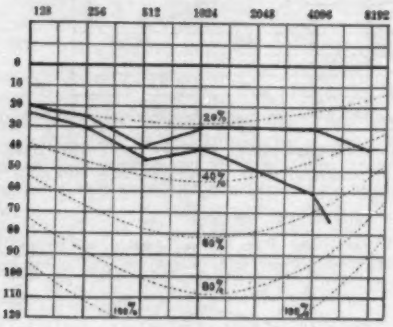


Figure 9

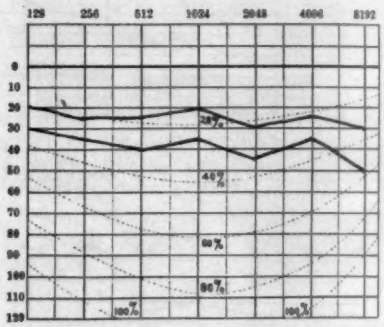


Figure 10

Fig. 9.—The lower line shows acuity for air-conducted sound before inflation, and the upper line, the improvement, particularly in the high frequency range, after inflation.

Fig. 10.—The lower line shows the acuity for air-conducted sound in a patient with perforated drum membrane, and the upper line, the improvement due to the application of a wet cotton tampon.

is functional the acuity is greater when the dehiscence in the drum membrane is occluded. The following case illustrates what is meant.

The patient had been practically deaf on the left side for years. Later purulent otitis media developed on the right side, which left her with a large perforation in the posterior quadrant of the drum membrane. The hearing acuity is indicated in the lower line of figure 10. The perforation was covered with a small tampon of wet cotton, with the result shown in the upper line. The increase in the acuity for air-conducted sound is fairly uniform throughout the frequency range. This result, however, is not characteristic for the condition in which the drum membrane and outer ossicles are wanting and the familiar cotton pellet prosthesis is applied.

Lillie and Kranz [13] reported the first quantitative experiments on the effect of the cotton pellet prosthesis in a patient who had had bilateral radical mastoidectomy. They observed that while the effective application of the pellet prosthesis increased the air conduction acuity for low frequencies by some 10 decibels, the sen-

13. Lillie, H. I., and Kranz, F. W.: Ann. Otol., Rhin. & Laryng. **33**:458, 1924.

sitivity for high frequencies was increased by a factor of about 30 decibels. This greater efficiency of the cotton pellet prosthesis when applied directly to the medial tympanic wall appears to be characteristic at high frequencies.

Some five years after the operation the patient previously mentioned in this paper who had been subjected to bilateral ossiculectomy was again examined. She had found that the application of a cotton pellet prosthesis was helpful. A series of tests was made on three occasions,[10] and the acuities with and without the prosthesis were averaged. The results are shown in figure 11; the greater improvement in sensitivity for high frequencies, while not as pronounced as that in the case of Lillie and Kranz, is evident. A similar result has been noted in a number of cases encountered during the past five years.

Opinions differ concerning how the cotton pellet produces the increase in air conduction acuity, as well as with respect to the contact area in the medial tympanic wall essential to the effect. Hughson, for example, expressed the belief that application of the pellet to the round window decreases the vibratory reactions in the secondary tympanic membrane and increases the acuity through immobilization of the round window membrane. I opposed this interpretation and suggested that the pellet vibrates to air-conducted sounds and acts as an inefficient transformer. The problem has been discussed in detail elsewhere.[14] Some investigators hold that the pellet must be applied to the area of the stapes; others believe that contact with the round window is essential, and still others feel that contact with the promontory is also effective. In any event, the proper area for an effective contact is critical, and this explains why the patient is usually more adept at a proper placement than is the otologist. The differences in opinion on the effective contact area will be discussed later in this paper.

Some twenty years ago I [15] published the results of an investigation on the sound conduction apparatus in birds. The bird's middle ear is quite unlike that of the mammal in that the ossicular chain is represented by a simple sticklike structure, known as the columella. Inasmuch as birds hear as acutely as mammals, and perhaps even more so, it was assumed that this simple sound conduction apparatus must be efficient. The correlation between the structure of the sound conduction apparatus of the bird and that of the mammal led to the idea that each acted as a resistance-matching transformer and that each represented a diaphragm and rod, an interpretation which was not enthusiastically received.[16] Inasmuch as a prosthesis patterned after the middle ear apparatus of the mammal would be extremely difficult to construct and to introduce, the possibility of replacing the lost sound conduction system in the human being with a unit patterned after the bird's ear presented itself. It was not until a number of years later that the opportunity came to make the trial.

In 1934 a patient reported for examination, indicating that he was prepared to submit to any series of experiments if this might lead to more acute hearing. He had had a radical mastoidectomy on both sides. The left external auditory canal was almost completely collapsed, but the right canal had been widely excavated. The medial tympanic wall could be inspected directly without a speculum. The patient was wearing a small cotton pellet prosthesis applied to a depressed area in the position of the oval window. The curve for air conduction acuity without and with the pellet prosthesis is shown in figure 12. It will be noted that the pellet prosthesis was not particularly effective, although the conferred acuity for high frequencies was somewhat greater than that for low frequencies.

14. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **45**:351, 1936.
15. Pohlman, A. G.: J. Morphol. **35**:229, 1921.
16. Pohlman, A. G.: Ann. Otol., Rhin. & Laryng. **31**:1, 1922.

A telephone probe similar to that described by Pohlman and Kranz [17] was constructed by affixing a long slender rod of bamboo to the vibrating button of a miniature bone receiver. The stem was tipped with a small ball of dental stent. The patient introduced the probe himself to make contact with the oval window area, and an acuity curve was obtained. Next, the probe was placed in contact with an upper tooth, and a second test was made. Under the first condition the probe operated like a sound conduction apparatus, and under the second what was heard were vibrations of the head as a whole, or bone-conducted sound. The information derived from these two tests is shown in figure 13, in which the upper line represents the sensitivity with the probe in contact with the oval window and the lower line the bone conduction sensitivity at the probe source. The two curves are fairly similar in form, and the increase in intensity required to hear from the tooth over that required when contact was made with the oval window averaged about 40 decibels for the frequency range from 512 to 4096 inclusive. In other words, it required 10,000 times the intensity to hear when the labyrinth liquid was set in vibration through the head as when it was set in vibration directly.[18]

A similar test was made with a telephone probe on a patient only a few months ago. In this patient both the oval and the round window were accessible to contact,

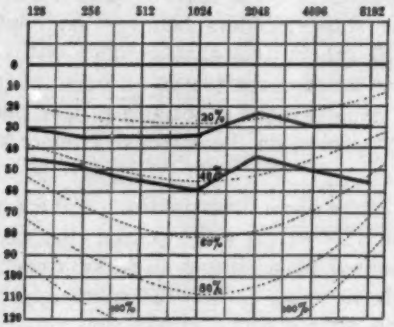


Figure 11

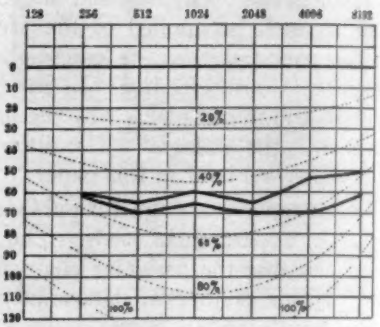


Figure 12

Fig. 11.—The lower line indicates the acuity for air-conducted sound without a cotton pellet prosthesis, and the upper line, the improvement with the prosthesis.

Fig. 12.—Graph showing the amount of improvement after radical mastoid operation through use of a cotton pellet prosthesis.

and the acuity with a telephone probe was tested through both windows and was compared with that obtained through contact with the promontory and with an upper tooth. The information thus obtained, which is shown in figure 14, indicated that both windows yielded equally, while the sensitivity through the promontory was somewhat less and the increase in intensity required from contact with the tooth was considerably greater. The fact that both windows in this patient were equally yielding is held as evidence against the accepted interpretation of mass movement reactions in the cochlea and is considered elsewhere.[19]

A simple imitation of the bird's sound conduction apparatus was constructed for the first experiment in the following manner: The two ends of a 00 gelatin capsule were punched out with a heated cork borer and the two parts of the capsule separated; a bit of a moistened fish skin condom was press-fitted into the larger

17. Pohlman, A. G., and Kranz, F. W.: Proc. Soc. Exper. Biol. & Med. **25**:504, 1928.
18. Pohlman, A. G.: J. Acoustic. Soc. America **8**:112, 1926.
19. Pohlman, A. G.: Further Objections to the Accepted Interpretations of Cochlear Mechanics, Arch. Otolaryng. **35**:613 (April) 1942.

segment of the capsule, the smaller segment being employed as a plunger, and the overhang of the smaller segment was cut away. When dry, the diaphragm thus produced was shellacked to make it somewhat stiffer and moisture proof. The columella of the bird was imitated by melting a small bead of dental stent on the end of a stout boar bristle. The free end of the bristle was fed through a hole pricked into the diaphragm with a needle, so that the ball tip was at a level with the opening in the capsule (fig. 15). The capsule was seated into the bony external canal, and the bristle was gently pushed into contact with the predetermined yielding area. When this was effected, the result was almost startling. The patient declared he could hear some one talking in a neighboring room but complained that the device was noisy. The noise proved to be the roar of a bunsen burner on the work table and disappeared when the burner was turned off. A simple test with the burner was made. Without the prosthesis the patient could just detect the noise at about 25 cm. from the ear, and with the prosthesis he could tell whether the burner was turned high or low at a distance of 7 meters. A comparison of the effectiveness of the cotton pellet and that of the diaphragm-rod prosthesis is shown in figure 16. It will be noted that the cotton pellet enhanced the high frequencies somewhat more than the low frequencies and that the same effect was obtained with the diaphragm-

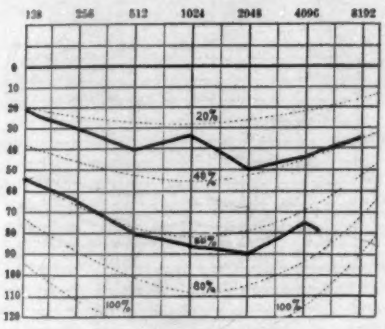


Figure 13

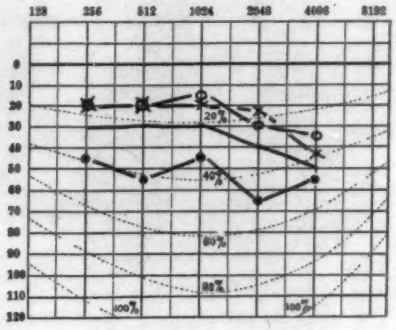


Figure 14

Fig. 13.—The upper line shows the sensitivity to a telephone probe applied to the oval window in the patient whose audiogram is shown in figure 12, and the lower line, the bone conduction acuity when the same probe was applied to a tooth.

Fig. 14.—Acuity determined through a telephone probe applied to the oval window (line with circles), the round window (line with crosses), the promontory (broken line) and the tooth (line with dots)).

rod insert. It is also evident that the improvement for frequencies of 512 to 4096 with the diaphragm-rod prosthesis over that with the cotton pellet was by a factor of some 15 decibels. The patient learned to make and insert the device himself. He prefers a loose coupling between the rod and the diaphragm, although the sealing of the diaphragm to the rod produced greater enhancement. He says that when he is disturbed by office noises he can withdraw the rod from contact with the oval window and go back to the original condition of deafness. Then, when he requires to hear, he pushes the rod back into position. This patient has worn the device effectively for about five years. The success in this and in a few other cases encouraged me to confine my attention to the problem, and certain technical improvements have been made.

It is obvious from this brief account that the contact area essential to the proper application of a diaphragm-rod prosthesis is extremely critical. This made it necessary to determine accurately the location of the area, and this problem was

solved through the use of a simple acoustic probe because of the danger and the difficulty in applying a telephone probe. The acoustic probe consists of a stalked diaphragm of mica which supports a slender stem of bamboo or of wire suitably tipped with a small ball termination (fig. 17). The probe is held by the stalk, and the stem is introduced through a speculum. The exposed medial tympanic wall is explored by touching it lightly here and there while the examiner keeps talking. When a yielding area related to the vestibular space is contacted, the patient will immediately declare the examiner's voice has suddenly become loud and deformed. The loudness is due to the vibrations of the mica diaphragm delivered through the stem. The deformation of the voice is due to the low free period of reaction in the probe. A positive effect is always elicited when contact is made with the movable oval window, or with the round window when this area is accessible. A negative effect obtains when contact is made with a fixed stapes or with the promontory.

This answers the question regarding the effective contact for the cotton pellet prosthesis. The result is negative if the contact is with a fixed stapes or with the promontory and is positive if it is with either of the two windows when the stapes is movable and when the round window is accessible—which is not often the case. The establishment of the location of the proper contact area by means of an acoustic probe does not mean that the cotton pellet prosthesis is always effec-

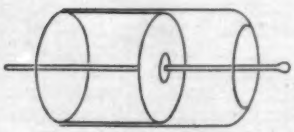


Figure 15

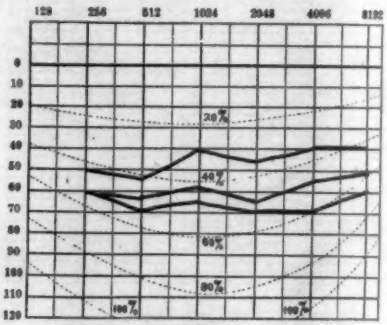


Figure 16

Fig. 15.—Diagram showing 00 gelatin capsule with press-fitted diaphragm of fish skin. The diaphragm is varnished when dry and a paper disk affixed to support the nylon rod. This insert is a temporary one and may be employed for purposes of testing.

Fig. 16.—The lower line shows the air conduction acuity without prosthesis in the patient whose audiogram appears in figure 13; the intermediate line, the effect of a cotton pellet prosthesis, and the uppermost line, the effect of the diaphragm-rod prosthesis.

tive when applied to this spot. Much seems to depend on whether the contact of the prosthesis can be confined to the restricted area. This explains why the patient is usually able to find the proper spot and to determine the size and form of the pellet to be used better than the otologist.

The diaphragm-rod insert has been improved in several ways. The gelatin capsule has been replaced by an aluminum cylinder, which must be accurately fitted into the bony external canal. The fish skin diaphragm is now treated with cellulose cement to make it waterproof, and a tiny paper disk is affixed to the diaphragm to prevent cutting when the rod is introduced. The boar bristle tipped with dental stent has been replaced by an insoluble, flexible rod of nylon 0.002 to 0.0022 inch (0.005 to 0.0056 mm.) in diameter. A ball tip may be made for the rod by heating the end carefully at an alcohol flame, and this ball tip may be flattened or cupped to meet the conditions at the contact area. Figure 18 indicates the result

when the air conduction acuity in 10 cases was averaged to form the lower line. In some of these cases the patient did not hear the highest frequency of 8192 at the maximum output of the audiometer, and the range has, accordingly, been restricted to frequencies of 128 to 4096 Hertz inclusive. It will be noted that the averaged air conduction acuity in these cases in which the drum membrane and the outer ossicles are wanting conforms with that for typical conduction deafness—a more or less uniform loss in sensitivity. The upper line shows the averaged increase in sensitivity through introduction of the diaphragm-rod prosthesis. The curve indicates improvement of about 10 decibels at frequencies of 128 and 256, while in the important range, from 512 to 4096 Hertz, the sensitivity is increased by practically 30 decibels. This result, to state it another way, is better than that usually afforded by the best quality of vacuum tube hearing aid.

The proper fitting of the diaphragm-rod prosthesis is, however, not as simple as it may appear. It is necessary to seat the molded metal capsule into the bony external canal medial to all conferred motion from the mandible, and the rod must be sufficiently short to make proper contact and not to project beyond the outer limit of the capsule. These requirements make it difficult to apply the prosthesis

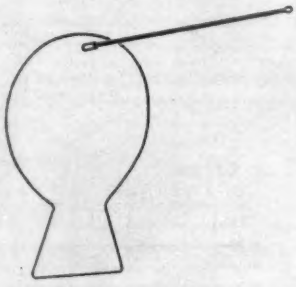


Figure 17

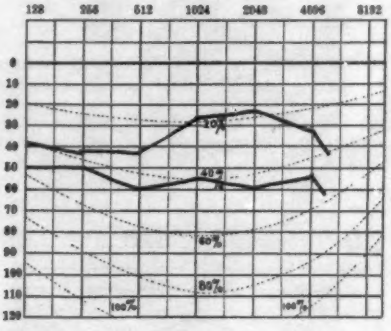


Figure 18

Fig. 17.—Stalked mica diaphragm supporting a wire probe tipped at the end for contact with the medial tympanic wall. The probe has been improved by its being made of metal and can be sterilized by boiling.

Fig. 18.—The lower line shows the averaged hearing acuity in 10 cases in which the middle ear apparatus had been lost or removed. The upper line indicates the averaged greater acuity after introduction of the diaphragm-rod prosthesis.

in contracted or narrow canals, especially in those in which the cartilage of the helix overhangs the external orifice. This may be illustrated in the case of a patient with bilateral conduction deafness due to radical operation. The external canals were sufficiently wide to accept the prosthesis, but the overhang of the cartilage made it impossible for the patient to adjust the rod herself. The acuity curves before and after introduction of the prosthesis are shown in figure 19, and the subjective effect of the useful and balanced hearing on the two sides was even greater than this degree of improvement would lead one to expect. It will be necessary in this case to remove the overhang of cartilage, and it is hoped that the patient will agree to this simple operation.

It is also extremely difficult to fit a prosthesis in cases in which the external canal has been widely excavated through removal of too much of the posterior wall and in which the external orifice is considerably smaller than the cavity into which the prosthesis must be placed. It is essential that these two points be borne

in mind when a radical operation is indicated and the possibility of a diaphragm-rod prosthesis considered.

The conferred acuity is dependent not only on the size of the diaphragm but on the character of the contact area. In this connection the results in 2 cases may be conpared. The first case was one in which there was complete conduction deafness on one side and a radical mastoidectomy had been performed on the other. The canal was widely excavated but was, fortunately, somewhat conical, with the external orifice much larger than the bony canal proper. There appeared to be complete resorption of the stapes, and the medial tympanic wall was heavily epidermized. The hearing acuity on this side is shown in the lower line of figure

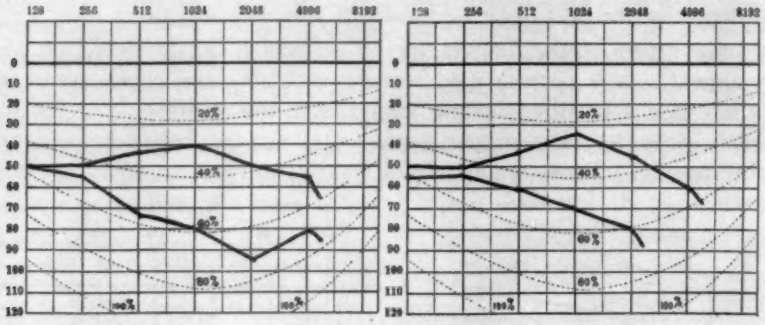


Fig. 19.—The lower lines present the air conduction acuity without prosthesis, and the upper lines, the increased acuity due to the diaphragm-rod prosthesis.

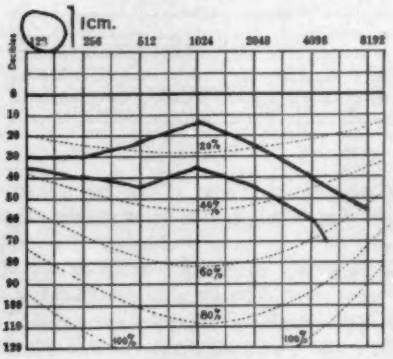


Figure 20

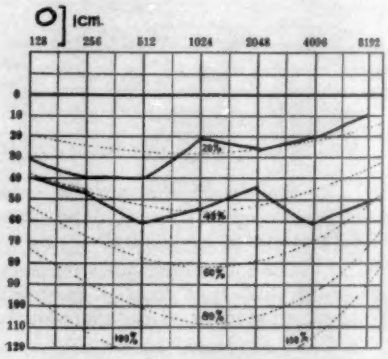


Figure 21

Fig. 20.—Improvement in air conduction acuity due to the diaphragm-rod prosthesis. The size of the diaphragm is indicated in the upper left corner.

Fig. 21.—Improvement in air conduction acuity due to the diaphragm-rod prosthesis. The size of the diaphragm is indicated in the upper left corner.

20, in which the size of the diaphragm employed is indicated in the upper left corner. The conferred acuity is shown in the upper line and is more or less uniform throughout the range tested. The second case is that of a young woman who had a radical operation. The bony canal had not been excavated and was relatively small and somewhat oblique. The epithelial covering of the medial tympanic wall was extremely thin, and the foot plate of the stapes seated in the oval window was readily defined. The patient accepted a prosthesis at the point where the size of the diaphragm is indicated in the upper left corner of figure 21. The

enhanced acuity in this case was pronounced, about 30 decibels, especially in the higher frequency range. Whether the difference in the amount of increased intensity is dependent on the size of the diaphragm or on the character of the contact area or on both has not yet been definitely determined.

The diaphragm-rod prosthesis, like the cotton pellet, is invisible, has no marked peaks of response and is nondirectional, and the efficiency even exceeds that of a vacuum tube hearing aid in favorable cases. There appears to be another advantage in the filtering effect on low frequency vibrations and in the obvious deformation of the acuity curve, which is, to all intents and purposes, the reverse of the characteristic curve for perception deafness when the middle ear apparatus is normal. Seemingly, the prosthesis cuts down adventitious noises and confers greatest improvement in the consonant range, which makes for intelligibility of spoken language. As evidence for this statement the following case is presented briefly. The patient had lost the drum membrane and ossicles on both sides, and only on one side could proper contact be effected. The acuity curve for air-conducted sound without prosthesis is shown in the lower line of figure 22A, and the improvement is indicated in the upper line. This was very acceptable to the patient. Some time later he reported that while the prosthesis was working, it was noisy

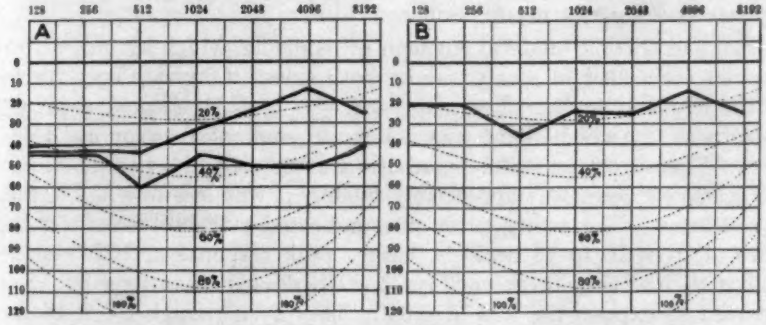


Fig. 22.—The upper line in *A* shows the effect of the diaphragm-rod prosthesis, and the line in *B*, the effect on the prosthesis due to loading with secretion.

and had lost its efficiency. An acuity curve was taken, and this is indicated in the solid line in figure 22*B*. It was found that there had been some discharge, which loaded the diaphragm, and when this was removed the prosthesis was quiet and the acuity returned to the former level.

While the purpose of a diaphragm-rod prosthesis was to replace the function of the normal drum membrane and ossicular chain, this has, fortunately, not been realized. It has been noted that the acuity curve presents a marked deformation as compared with the normal. This deformation results in a filtering effect on the low frequency vibrations and in a maximum improvement in the range important to speech and music, 512 to 4096 double vibrations. In a general way, the hearing curve for a patient with a normal internal ear who wears a prosthesis is practically the reverse of that for one with a normal middle ear apparatus and the usual perception deafness. A patient with perception deafness usually indicates that spoken language is more or less unintelligible, that loud talking increases the disability and that loud noises disturb him seriously. A patient with complete conduction deafness due to a nonfunctional but intact middle ear, just as the person in whom the middle ear apparatus is wanting, tells a different story. He indicates that while conversation may not be heard intelligibly, he does hear loud talking distinctly and that he appears to hear more acutely under conditions of noise. The lack of

intelligibility of spoken language in both the perception-deafened and the conduction-deafened patient is due to a relatively lowered acuity for consonant sounds. In the patient with perception deafness, however, with excellent hearing for low tones, intelligibility is seriously masked by the adventitious noises, while for the patient with conduction deafness it is not. Further, when speech is loud the perception-deafened patient may tend to damp the sound conduction apparatus through contraction of the stapedius muscle and superimpose a physiologic conduction deafness on the already present lowered sensitivity of the internal ear. The conduction-deafened patient, on the contrary, does not have this protective reflex through the stapedius muscle.

The patient wearing a diaphragm-rod prosthesis maintains the conduction deafness for low frequencies, which reduces the masking effect of noises and exaggerates the sensitivity with which the consonant sounds are heard. The result is that the deformation in conduction appears to produce a disproportionate increase in the intelligibility for spoken language. In other words, the normal person would probably hear spoken language even more intelligibly if he were less sensitive than he already is to air vibrations of low frequency.

There are exceptions to the rule that the cotton pellet prosthesis tends to produce greater sensitivity to air sounds for high tones than for low tones, and the following case is unique in my experience.

A woman had chronic bilateral purulent otitis media, which left her with remains of the ossicles embedded in scar tissue on both sides. One side only reacted positively to the acoustic probe. The acuity curve (fig. 23) showed a peculiar conduction deafness, in which there was a definite peak of reaction to 2048 double vibrations. When a cotton pellet prosthesis was applied, the patient declared that while the speaker's voice was appreciably louder, it was less intelligible than without the prosthesis. The acuity curve now indicated a reenforcement to the lower frequencies of from 10 to 15 decibels, with a peak response at 1024 double vibrations, and with lowering of the acuity at 2048 double vibrations by 15 decibels. This appeared to explain the loss of intelligibility even though the loudness of the voice was increased. In other words, the patient who showed a deformation in the curve for air conduction acuity similar to that brought about by a diaphragm-rod prosthesis heard spoken language more intelligibly than when the application of the cotton pellet prosthesis brought about a less deformed reception.

A case was mentioned earlier in this paper in which excision of a segment of the drum membrane improved the acuity for air vibrations. An acoustic probe was introduced, and the stapes was contacted. The effect was positive. Next, a temporary diaphragm-rod insert was placed in the external auditory canal, and effective contact was made at the stapes. The effect was dramatic, and the acuity curves with and without the prosthesis are shown in figure 24. The prosthesis had little effect at low frequencies (5 decibels), but for the range of 512 to 4096 double vibrations the acuity was improved by a factor of 35 decibels.

A second case is also interesting. An elderly woman had lost the middle ear apparatus on both sides many years ago; the tympanic cavities were dry, and no yielding area could be detected with an acoustic probe. The patient was so disappointed that nothing could be done that an attempt was made to soak the dry surface on the medial tympanic wall. A pellet of glycerin diluted with a solution of boric acid was introduced, which was renewed daily by the patient. A week later the acoustic probe showed a positive result. The acuity curve without a diaphragm-rod prosthesis (fig. 25) indicates pronounced perception deafness. The effect of the prosthesis, shown in the upper line, represents an improvement of some 30 decibels.

While there are no indications for ossiculectomy in cases of complete conduction deafness with intact drum membranes, operation has been performed in several cases of this type of deafness with dehiscence of the drum membrane and discharging ears. A report on these cases must be deferred until the experimental evidence has been completed.

The results thus far are gratifying and support the idea that a sound conduction apparatus is important to acute hearing for all frequencies. However, Nasiell,[3] in a recent report on extensive experiments with a cellophane prosthesis, indicated confirmation of the interpretation of dual conduction. He stated:

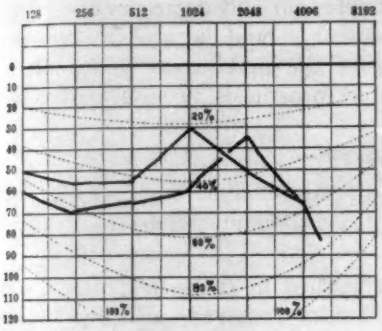


Figure 23

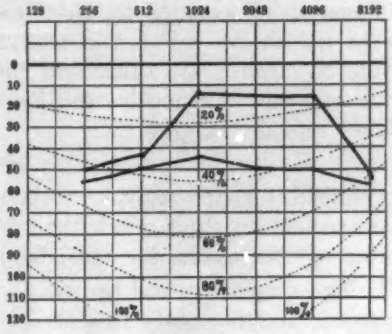


Figure 24

Fig. 23.—The upper line, with a peak at a frequency of 1024, shows evidences of increased sensitivity to low tones after introduction of a cotton pellet prosthesis.

Fig. 24.—Graph presenting a further observation on the patient whose hearing acuity is shown in figure 5, when the rod of the diaphragm-rod prosthesis was introduced through the incision in the drum membrane.

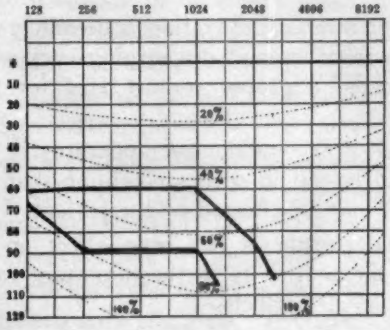


Fig. 25.—Effect of a diaphragm-rod prosthesis on a patient with profound perception deafness.

It is therefore established that the ossicular chain is most important for the conduction of low tones and the intermediate range of frequencies, while high tones are conducted principally through the air chamber of the middle ear—the acoustic capsule of the tympanum—and the perception of high tones suffers little reduction through loss of the drum membrane and ossicles.

It is unfortunate that Nasiell did not give the acuity curves before and after the introduction of the prosthesis, and, from my results in the single case reported in this paper, it would be unfortunate, so far as intelligibility for spoken language is concerned, if Nasiell was correct. The idea that the middle ear acts as an acoustic capsule is not, in my opinion, correct. The effective operation of an

acoustic capsule is dependent on a relatively large diaphragm and a relatively small amount of trapped gas. The device would not be effective with a small drum membrane and a large amount of air trapped in the middle ear. The introduction of a capsule provided with a diaphragm has shown no significant effect in any of the cases in which the middle ear apparatus is wanting. Probably in time an incision of the drum membrane may be indicated in order to determine whether or not a proper contact area for a prosthesis is present, before the nonfunctional middle ear apparatus is removed.

While the prosthesis has been worn continuously for as long as four to six weeks without signs of irritation, this practice cannot be recommended. The accumulation of secretion tends to load the diaphragm and decreases the effectiveness of the device. For this reason, the patient should be able to remove the prosthesis, cleanse the canal and the cavity of the middle ear and replace the device himself. Obviously, the diaphragm-rod prosthesis is contraindicated for patients with discharging ears.

CONCLUSIONS

While this paper reports the results of several years of work, it may not be considered as anything more than a preliminary communication; nevertheless, a number of conclusions are indicated.

1. The diaphragm-rod prosthesis may be applied in cases in which the drum membrane and the outer ossicles have been lost or removed, provided the ear is dry, the external canal normally wide and the proper contact area for the rod accessible.

2. This contact area must be sufficiently yielding to accept the vibrations of the prosthesis—a condition which may be readily established with the acoustic probe.

3. Useful hearing may be conferred on practically all patients under the conditions noted in 1 and 2.

4. An ossiculectomy or a radical operation is definitely indicated for all patients with perforated drum membranes and discharging ears whose condition does not yield to treatment, provided a proper contact area for the prosthesis has been established.

5. When the removal of the middle ear apparatus is indicated, the bony and cartilaginous canal should be left in such a condition that proper placement of the diaphragm-rod prosthesis may be effected by the patient.

6. The diaphragm-rod prosthesis has little effect on the sensitivity for low tones and may increase the sensitivity in the important range of frequencies from 512 to 4096 by as much as 30 decibels.

7. On an average, the efficiency of the diaphragm-rod prosthesis is at least 15 decibels greater than that of a cotton pellet, and it may be applied in cases in which the cotton pellet is of no benefit.

8. The effect of both the cotton pellet and the diaphragm-rod prosthesis demonstrates a greater increase in air conduction acuity for high frequencies and opposes the interpretation of a dual conduction and of the round window route as effective in the conduction of air vibrations to the cochlea.

9. The diaphragm-rod prosthesis is contraindicated for patients with discharging ears.

4056 Farmouth Drive.

SYMPTOMATIC AND EMPIRIC TREATMENT OF THE ALLERGIC NOSE

FOLLOW-UP QUESTIONNAIRE IN CASES IN WHICH RHINITIS WAS REFRACTORY TO TREATMENT

EDWARD KING, M.D.
AND
J. HAROLD KING, M.D.
CINCINNATI

The theory that vasomotor rhinitis or allergic rhinitis is of nasal origin had its inception about 1880. The explanation given for this theory throughout many years was that there were sensitive areas in the nose which had to be eradicated in order to relieve the symptoms. Accordingly, a host of remedies was developed, each of which was declared to have more or less merit in accomplishing this end. Most physicians are familiar with the various measures recommended, such as cauterization, coagulation, injections, ionization, and so on.

Advances in the field of allergy, together with a better understanding of the physiology of the nasal mucosa, contributed most to the gradual elimination of therapy which was based on a false assumption. Along with the allergists, the rhinologists are making some progress in the understanding and treatment of these conditions. They are recognizing an increasing number of patients in their daily practice complaining of nasal symptoms that are due to an allergic state. It has been estimated that in at least one third of all patients manifesting nasal symptoms these are allergic in origin. To me it seems that the estimate is low.

Some rhinologists are qualified to do the cutaneous tests and carry out the treatment. However, the rhinologists must keep in mind that the general physical condition of the patient is important when nasal symptoms are being evaluated. A general physical examination should be done on these patients, otherwise the rhinologist may be discovered treating cardiac asthma with allergens. A cooperative spirit in studying these cases is most desirable and more beneficial to the patients.

Every patient complaining of nasal symptoms uses some form of shrinking solution or vapor. It is my experience that these remedies are overused and harmful to some patients. In a good many instances the discontinuance of the shrinking medication has cleared up the symptoms. It is my practice to ask the patient on his first visit to give up all

Read at the meeting of the Ohio State Medical Association in Columbus, Ohio, April 30, 1942.

drops and inhalers for a few days. Judicious use of cocaine has been valuable in extremely uncomfortable patients. A small amount of a 0.5 per cent solution of a cocaine salt is prescribed and the patients instructed to apply the solution with a cotton applicator. Cocaine substitutes have never been successful in my hands.

Spurs and deviations of the septum must receive consideration in the treatment. It is my observation that symptoms of allergy occur in patients who have little other nasal abnormality. If a septal deviation is present, the history will disclose whether obstruction was present previous to the onset of symptoms. If nasal polyps are present, they should be removed before any other procedure is followed.

Certainly every physician wants to know whether a patient with an allergic nasal condition or asthma has a sinal infection. There are a good many pitfalls in the diagnosis of sinal disease in the allergic patient. The nasal smear should be studied frequently. The history must be detailed in reference to colds and attacks of sinal disease.

Transillumination and roentgen examination must be repeated frequently in order to rule out allergic swelling as the cause of cloudiness. There is no doubt that a polypoid membrane may be present in purely infectious sinusitis. One can prove this assertion by the large polypoid masses which are found in the nasal passages and in the antrum following infection through an abscessed tooth. There is hardly any doubt in my mind that hyperplastic ethmoiditis and polypoid swelling of the nasal mucosa is allergic in the majority of cases. At times all have seen cases in which the sinal infection is secondary to the allergic swelling. The use of iodized oil in diagnosis and treatment is of much value. Gentle treatment of the nose is highly desirable. Sometimes irrigation of the sinuses must be resorted to when large accumulations of mucus and pus are present. Only rarely is it necessary to adopt procedures in which the entire mucosa of the sinuses are eradicated. It is best to make it clear to the patient that the operative procedure is undertaken with the hope of establishing free drainage while the underlying cause may remain a mystery.

In spite of every measure adopted and every effort put forth by the allergist and the rhinologist, there are patients whose disease is refractory to treatment. They offer a problem in diagnosis and treatment. A few of those who have come under my care recently may be of some interest. A brief review will be given of the cases of 9 patients. They belong to a group whose rhinitis did not respond to treatment, and all of them complained of the same nasal symptoms. In all of them the cutaneous tests were negative. The following case is typical:

W. Z., aged 46, came to the office on Nov. 12, 1940, complaining that his nose was stopped up. He stated that the trouble began the previous July, while he was on a business trip, and that he was told by the physician who treated him

that he had a cold; he improved with treatment. Three weeks prior to examination by me, while he was on a business trip in Indiana, his nose became stopped up and he had sneezing spells. The difficulty became gradually worse, and for the past week he had been unable to get the nose open in spite of every measure. The discharge was watery and colorless. He had no headache and stated that he had no fever at the onset. He had never been subject to colds or sore throat and had never had any trouble with the sinuses. He did have an operation on the nasal septum when he was 16 years old because of an injury, and after that he experienced no trouble with breathing. His past history showed that he had no allergy, and there was no family history of allergy.

The nasal mucosa was pale and the septum deviated to the left. It was not possible to make a postnasal examination. The tonsils were submerged and contained quite a little secretion. The transillumination showed both maxillary antrums and the right frontal sinus cloudy. Roentgenograms showed clouding of the right frontal sinus and the right antrum. A nasal smear was made and showed mostly neutrophils, with many eosinophils. On irrigation of the right antrum, some thin yellow pus was obtained. A small amount of a 1 per cent solution of a cocaine salt was prescribed, together with some capsules containing acetylsalicylic acid, phenobarbital, ephedrine and a very small amount of antimony and potassium tartrate, and the patient was advised to return in a week. The treatment consisted of shrinking the nose with cocaine and irrigation of the antrum. His condition improved somewhat under this treatment, although the nose swelled tight at times and an intractable cough developed. At this time he was referred to the allergist (Dr. King), who reported reactions to all cutaneous tests negative. The Wassermann test was negative. Local treatment to the nose was continued, and the administration of stock vaccine was begun by the allergist, together with injections of dust.

During the last three months of the period of observation this patient was seen about every two weeks. During that time trial diets were instituted, histaminase therapy (torantil) was tried and thyroid was given; insulin also had a trial. Mild ionization of the mucosa on the right side was done as a last resort. All of these measures were without any apparent benefit to the patient. During the latter part of his treatment, nasal smears showed all cells eosinophils. It was finally decided to operate on the right antrum because of persistent swelling of the mucosa, which was disclosed by roentgen rays and iodized oil. The patient did not return for the operation.

The next case shows the influence of the endocrine system on the nasal mucous membrane. The patient had the same symptoms.

Mrs. H. M., aged 38, was first seen in September 1937, complaining of a cold and severe attacks of sneezing. She stated that she had been sneezing for seventeen years. Because the trouble began in August she was said to have hay fever. When it persisted through the years, however, she became quite sure that the diagnosis was wrong, especially since she had more trouble in the winter than in the summer. This patient had been subject to frequent colds, and with the colds she had a great deal of itching of the eyes and some itching in the roof of the mouth. She volunteered the information that the only time she had relief from sneezing was when she was pregnant. She had had four pregnancies. In 1928 she had undergone double ethmoidectomy with removal of the middle turbinates. Apparently she had not received any relief.

My examination showed the nasal mucosa red and the septum slightly deviated to the left. The middle turbinates had been removed. The sinuses appeared clear. She was referred to the allergist, who reported that she complained of

being cold most of the time and of dryness of the hair. She had begun to menstruate about the age of 13; at the time of examination she had not menstruated for two months. She was 25 pounds (11 Kg.) overweight.

The general physical examination showed nothing abnormal except a basal metabolic rate of —10 and a few signs of hypothyroidism. Cutaneous tests revealed that she was slightly reactive to dust, silk and cat dander. Thyroid substance was administered; she improved and her menstrual periods, which had been scanty, became more abundant. She continued to come for treatment of colds and stated that she felt better generally but that the sneezing persisted. During 1939, while visiting one of her children in the hospital she had a fainting spell. She was given a thorough physical examination. The physician who examined her prescribed an estrogen, and she stated that the sneezing disappeared immediately. She still has colds but less frequently, and no sneezing.

It is not my intention to give the details of each case. What can be learned about the group is sufficient for present purposes. A study of these cases as a whole reveals the following facts: 1. The chief complaint was the same: nasal symptoms consisting of obstruction, sneezing and watery discharge. 2. All patients said that the symptoms started with a cold. 3. The nasal mucosa was pale in 7 patients and red in 2. 4. The septum was deviated in 2. 5. Polyps were present in 2. 6. The postnasal examination showed no changes in 7 and polyps in 2. 7. The tonsils were submerged and infected in 5. 8. Roentgen findings in the sinuses were positive in 4 cases and negative in 5.

The cutaneous tests were negative in all cases, and all the patients had about the same nasal and general treatment. Three patients had nasal operations; 1, an operation on the nasal septum; 1, an operation on an antrum, with removal of mucosa on both sides, and 1, operations on both antrums and external operations on the frontal and ethmoid sinuses.

The 9 patients recently were sent the following questions: 1. Are the symptoms the same? 2. Is there any improvement? 3. What have you tried since last seen? 4. Have you any cough or asthma? 5. Have you found the cause of your symptoms?

Five patients answered: One noted improvement after moving to Baltimore; one experienced improvement without any treatment; one said improvement began with nasal treatment and injections of some iodine preparation; one reported improvement after complete removal of polyps and sinal mucosa, and the fifth, after using acetylsalicylic acid and mistol [1] drops.

SUMMARY

The cases of 9 patients have been analyzed because these patients all had the same symptoms, their reactions to cutaneous tests were negative and their disease was refractory to treatment. What conclusions may one draw from this analysis?

1. Mistol is liquid petrolatum containing camphor, eucalyptol, menthol esters and chlorobutanol.

After numerous discussions, my associate, Dr. J. Harold King, and I feel justified in the following recommendations: 1. Determine the skin reactions as soon as possible. 2. Control the environment. 3. Try diets strictly free from milk and wheat over extended periods. If there is no response, bacterial allergy must be considered. Cultures of the nose and sinuses can be made, and the administration of stock vaccines should be started. A trial should be made of the various empiric remedies, such as histaminase, thyroid and estrogens. Chemotherapy should be given in selected cases. A careful record should be kept of the procedures used and the length of trial of each. One hears of sudden cessation of symptoms in rare cases on the first visit through the use of a vaccine or some empiric remedy, but good results in the vast majority of cases will come only through careful painstaking effort with each patient.

Carew Tower.

STAPES, FISSULA ANTE FENESTRAM AND ASSOCIATED STRUCTURES IN MAN

IV. FROM FETUSES 75 TO 150 MM. IN LENGTH

BARRY J. ANSON, PH.D. (MED. SC.)
AND
EARL W. CAULDWELL, M.S.
CHICAGO

In continuing a comprehensive study of the stapedial region of the human ear, special attention has been given to the stapes, the vestibular window and the associated fissular tracts in developmentally important embryonic and fetal stages. In a preceding article[1] the steps in morphogenesis were traced as encountered in specimens from 6.7 to 50 mm. in crown-rump length; the current article carries the account through the 150 mm. specimen, at which stage the formation of bone is initiated in the stapes. Both of the contributions just referred to serve to fill hiatuses existing in two more general papers on stapedial, fissular and fenestral anatomy.[2]

MATERIAL AND METHODS

For this stage of the investigation reconstructions of the stapes, together with the surrounding area, were prepared by the wax plate method of Born.[3] Representative developmental stages were selected for reconstruction at approximately 25 mm. intervals, while comparable or additional anatomic features in intermediate stages were demonstrated by means of photomicrographs. All measurements were in crown-rump length.

Presented as part of an exhibit at the 1941 meeting of the Academy of Ophthalmology and Otolaryngology, in Chicago. Read at the 1942 (New York) meeting of the American Association of Anatomists, in New York (Anat. Rec. **82**:395 [March 25] 1942).

From the Department of Anatomy and the Department of Otolaryngology, Northwestern University Medical School. Contribution no. 383 from the Anatomical Laboratory.

Dr. T. H. Bast, of the department of anatomy of the University of Wisconsin, permitted us to study many of his excellent temporal bone series.

This investigation was conducted under the auspices of the Central Bureau of Research of the American Otological Society.

1. Cauldwell, E. W., and Anson, B. J.: Stapes, Fissula Ante Fenestram and Associated Structures in Man: III. From Embryos 6.7 to 50 Mm. in Length, Arch. Otolaryng. **36**:891-925 (Dec.) 1942.

2. Anson, B. J.; Karabin, J. E., and Martin, J.: (*a*) Stapes, Fissula Ante Fenestram and Associated Structures in Man: I. From the Embryo of Seven Weeks to That of Twenty-One Weeks, Arch. Otolaryng. **28**:676-697 (Nov.) 1938; (*b*) II. From the Fetus at Term to the Adult of Seventy, ibid. **29**:939-973 (June) 1939.

3. All reconstructions were prepared at a magnification of 125 diameters by the wax plate method from tracings made with an Edinger projection apparatus. The reconstructions of the otic capsule and the stapes include sections from the representative stages as follows: 75 mm., 43 sections; 78 mm., 64 sections; 100 mm., 62 sections; 111 mm., 66 sections; 126 mm., 78 sections; 150 mm., 90 sections. Sections of the 78 mm. series were cut at 18 microns; those of the 75 mm. series, at 200 microns, and all other series, at 25 microns.

Each reconstruction originally included not only the stapes but the capsular wall of the vestibular window and the adjacent fissula as well. For several of the drawings, these reconstructions were dismantled or cut, in order that either form or topographic relationships could be more advantageously recorded.

Except for the model of the fissula (126 mm.), the reconstructions represent solid tissues, namely, cartilage and bone. The fissular model (fig. 35), however, represents the fissular channel; it is essentially a "cast" of the intracartilaginous space of the fissula.

Reconstructions were prepared at the following stages:

Length, mm.	Age, Weeks *	Source	Catalogue No.	Figures
25	8	Wisconsin	87	1
50	11	Wisconsin	86	2
75	13	Northwestern		
78	13	Northwestern		3
100	15½	Wisconsin	22	6, 7
111	16½	Wisconsin	50	8, 9, 41, 42
126	18	Wisconsin	11	4, 5, 32, 33, 34, 35
150	20	Wisconsin	39	10, 11, 43, 44

Sections from the following stages are represented by photomicrographs:

Length, mm.	Catalogue No.	Series	Figure	Original Magnification
75	Northwestern	385 (R)	12	85
75	Northwestern	184 (L)	13	85
75	Northwestern	199 (L)	14	85
78	Northwestern	75 (R)	15	50
78	Northwestern	86 (R)	16	50
78	Northwestern	75 (R)	17	300
78	Northwestern	75 (R)	18	300
100	Wisconsin 22	16.2.3 (L)	19	23
100	Wisconsin 22	17.2.3 (L)	20	23
111	Wisconsin 50	16.1.0 (L)	23	85
111	Wisconsin 50	18.1 (L)	24	85
111	Wisconsin 50	19.3 (L)	25	85
111	Wisconsin 50	19.8 (L)	26	85
111	Wisconsin 50	20.3 (L)	27	85
111	Wisconsin 50	20.8 (L)	28	85
112	Wisconsin 53	23.1.2 (L)	21	50
112	Wisconsin 53	24.2.1 (L)	22	50
112	Wisconsin 53	24 (L)	22 a	50
126	Wisconsin 11	24 (L)	29	50
126	Wisconsin 11	25.2.4 (L)	30	50
126	Wisconsin 11	27.1.1 (L)	31	50
150	Wisconsin 39	20.5 (L)	36	85
150	Wisconsin 39	21.3 (L)	37	85
150	Wisconsin 39	21.8 (L)	38	85
150	Wisconsin 39	21.8 (L)	39	225
150	Wisconsin 39	25.5 (L)	40	85

*Ages, expressed in weeks, are based on computations employing the formula of Arey: crown-rump length, in centimeters × 0.3 = age in months, 1 month being added to the result for fetuses less than 10 cm. long.

OBSERVATIONS AND COMMENT

In the embryo of 25 mm. the stapes is composed of precartilaginous tissue; in general form it is ring shaped, not stirrup shaped (fig. 1); at the 30 mm. stage the annular form persists, but transformation into true cartilage, of hyaline type, has been accomplished.[4] At the 50 mm. stage the shape suggests that of a stirrup (fig. 2), while true stapedial form is attained in the 75 and the 78 mm. stage (fig. 3).

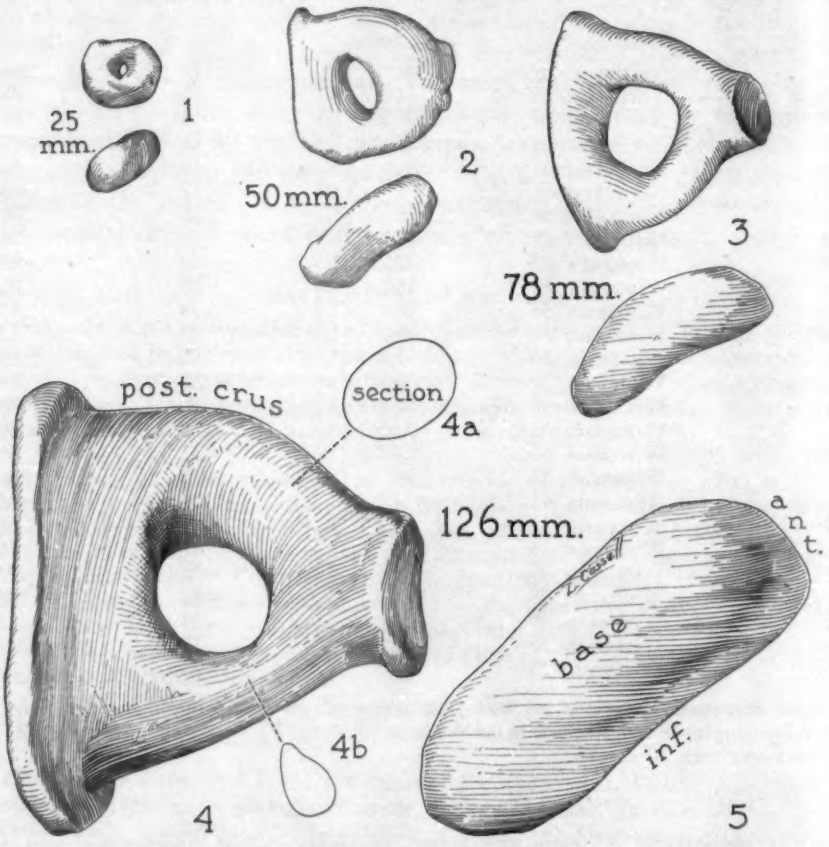
75 Mm. Fetus (13 Weeks).—In the 75 mm. fetus the otic capsule is composed wholly of mature hyaline cartilage, as are also the developing ossicles. Ossification is observed only in the basal lamina of the occipital bone connecting the apexes of the right and left capsules.

In the interval between the 50 and the 75 mm. stage the stapes undergoes modifications not only in size but in shape and relationships. At 50 mm. (fig. 2)

4. Descriptions of the 25, 30, 34, 43 and 50 stages appearing in the current article are dependent on the detailed descriptions of these specimens in a preceding paper.[2]

it presents a roughly stirrup-shaped appearance, with short, slightly bowed and relatively massive crura; a cylindric head, foveate on the incudal (articular) surface, and a base somewhat flattened on its vestibular side and rounded on its tympanic aspect. This stapes presents the first evidence of a basal rim, identifiable as a low ridge on the posterior and superior margins, while anteriorly and inferiorly a corresponding lip has not yet appeared. The obturator foramen is oval and relatively small.

When the fetus has attained a length of 75 mm., the characteristic stapedial form has been definitely attained, subsequent modification being in the directions of increase in size and alteration in histologic structure. The crura are distinctly



Figs. 1 to 5.—Reconstructions of the stapes; superior and medial views, in pairs. × 14.

Abbreviations appearing in these figures, and in the accompanying figures, are interpreted as follows: *Ann. lig.*, annular ligament; *Ant. crus*, anterior crus of stapes; *Cart. mass*, *Cart.*, cartilage (mature); *Coch.* or *C.*, cochlea; *F. a. f.*, fissula ante fenestram; *F. o.*, fenestral orifice of fissula ante fenestram; *Immat. cart.*, immature cartilage (young true cartilage); *I*, incus; *M*, malleus; *N. VII*, facial nerve; *Post. crus*, posterior crus of stapes; *Sc. V.*, scala vestibuli; *Stap.*, stapes; *Stap. m.*, stapedius muscle; *T. o.*, tympanic orifice of fissula ante fenestram; *T. t. m.*, tensor tympani muscle; *Tymp. cav.*, *T. C.*, tympanic cavity; *Tymp. memb.*, tympanic membrane; *Vest.*, *V.*, vestibule; *V o.*, vestibular orifice of fissula ante fenestram; *V. w.*, margin of vestibular (oval) window.

bowed and columnar, converging laterally to form an anatomic neck, on which is superimposed a slightly tapering columnar head; the foveate articular surface is better defined (fig. 3). The base is reniform. The width of the base is slightly

greater opposite the anterior crus than at the posterior crus, at which site the base is gently tapered. A narrow, rounded flange bounds the entire periphery of the base and is clearly distinguishable, histologically, from the deeply staining zone of condensed mesenchymal tissue which separates the basal rim from the fenestral margin of the capsule (fig. 12).

While growth changes are evidenced in all parts of the stapes, the cellular differentiations which characterize appositional growth particularly are seen to advantage in the region of the base. Here, the chondrogenetic activity of the basal perichondrium and of the primordial annular ligament maintains a cellular stratification as distinct as that observed in earlier stages (43 mm., 50 mm.); these zones are best represented in the 78 mm. stage (figs. 17 and 18) and will be fully discussed when that specimen is considered.

The fissula ante fenestram at the 50 mm. stage is little more than a short, wedge-shaped invagination of fibroblastic tissue into the capsular cartilage between the vestibule and the scala vestibuli, but at this stage a tympanic orifice is wanting. At 75 mm. the fissula ante fenestram not only is longer but communicates with the tympanic as well as with the vestibular cavity (figs. 13 and 14). Histologically, that type of cellular differentiation has begun which will characterize fissular structure throughout fetal and postnatal life. At the level of the tympanic orifice the proximal position (that nearest the orifice) of the fissula consists wholly of deeply stained fibroblastic tissue directly continuous with the cochlear perichondrium (fig. 13); however, the distal two thirds (anterior to the orifice) is represented by loose mesenchymal tissue of the most primitive sort—a reticular type resembling the periotic mesenchyme prior to vacuolation. The fissula extends caudally to a level of 0.38 mm, below the cranial extremity of the tympanic orifice, at which point it is continuous with the primordial annular ligament (fig. 14); the vestibular orifice then makes its appearance in the succeeding section, 0.02 mm. below the aforementioned level. This is the earliest stage at which we have encountered a continuous fissular channel (a vestibulotympanic cleft), extending from the tympanic orifice [5] through the annular ligament to the vestibular orifice (cf. 111 mm. stage).

A small stapedial artery courses through the obturator foramen of the right stapes (fig. 12); it arises in the vicinity of the facial nerve as a branch of the stylomastoid artery and becomes lost anterior to the stapes in the vascular mesenchyme of the tympanic cavity.[6] A recognizable stapedial artery was not in evidence in the series between the 30 and the 75 mm. stage.

78 Mm. Fetus (13 Weeks).—The stapes of the 78 mm. fetus differs from that of the 75 mm. fetus (its twin) in two respects: the over-all dimensions of the former specimen are slightly greater, the base less rounded and with less tendency to bulge into the vestibular cavity.

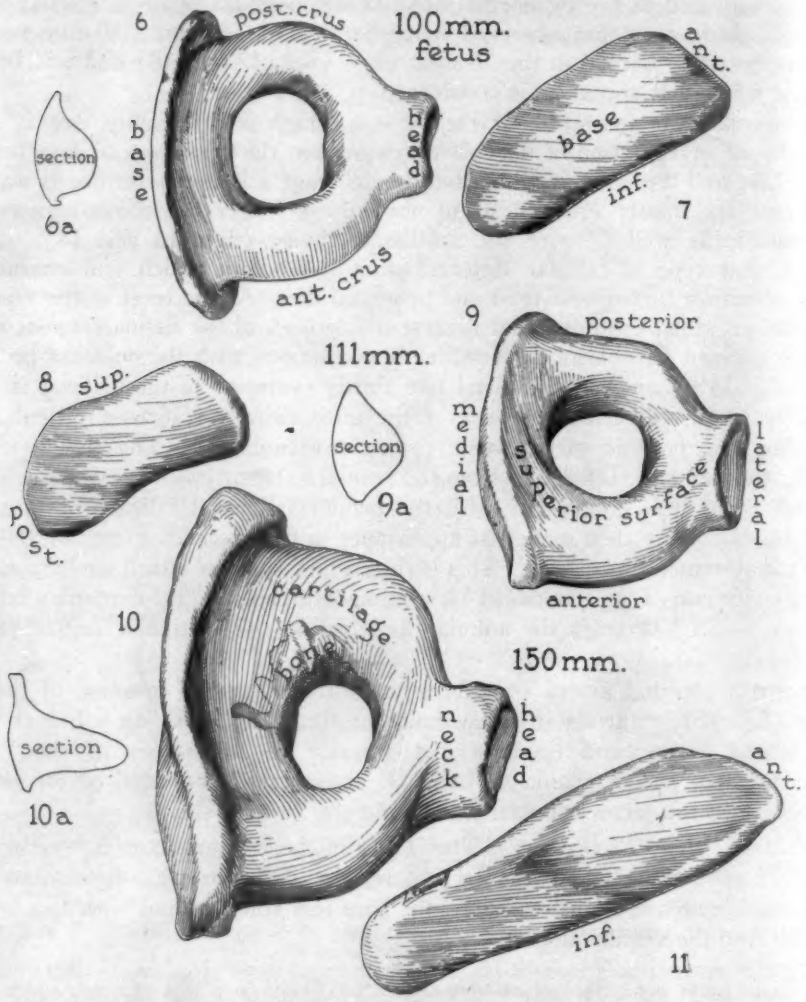
---

5. In an adult bone freed of all soft tissues the fissula is a true channel, which extends through the capsular wall from the tympanic to the vestibular cavity; in that form it was described by the original observers. Like the cochlear and vestibular aqueducts, the fissula extends from the cavity of the osseous labyrinth to an extracapsular space. In the fresh or preserved (uncleaned) bone the fissula is filled with connective tissue, which is continuous with periosteum at its vestibular and tympanic ends.

6. The term "tympanic cavity," as employed in these articles, refers to the space bounded by the tympanic membrane laterally and the lateral wall of the otic capsule medially; i. e., the name refers to the potential cavity of the fetus or the actual space in the adult. This usage makes for uniformity in discussion of the stapes, stapedial muscle, promontory and tympanic opening of the fissula ante fenestram and avoids the necessity of accounting for the changing relation of the mucous membrane to these structures of the midline ear in the embryo and fetus.

Stapedial growth is remarkable between the 50 and the 78 mm. stage; the length of the stapes has been exactly doubled and the length of the base increased by one third.[7] Another gross feature deserves comment: The crura are now of unequal length (fig. 3), the posterior crus being the longer of the two (0.76 and 0.61 mm., respectively). In general, a comparable linear difference in crural measurements characterizes the later stages.

The densely packed, deeply stained mesenchymal cells comprising the annular "ligament" form a distinct zone between the fenestral margin and the stapedial



Figs. 6 to 11.—Reconstructions of the stapes; superior and medial views; in pairs. ×8.5. Figures 6a, 9a and 10a represent cross sections through the bases of corresponding stapes.

7. By length of the stapes is meant the distance between the incudal surface of the head and the vestibular surface of the base measured in a straight line through the obturator foramen; were the stapes placed arbitrarily in the regular position of a stirrup, the measurement would be that of the vertical height. By length of the base is meant the greatest distance between the anterior and the posterior extremity of the base, measured in a line either coinciding with or running parallel to the long axis of the base. The width of the stapedial base is taken at a right angle to the length, usually at a point midway between the extremities; unless otherwise stated, it is the maximum width.

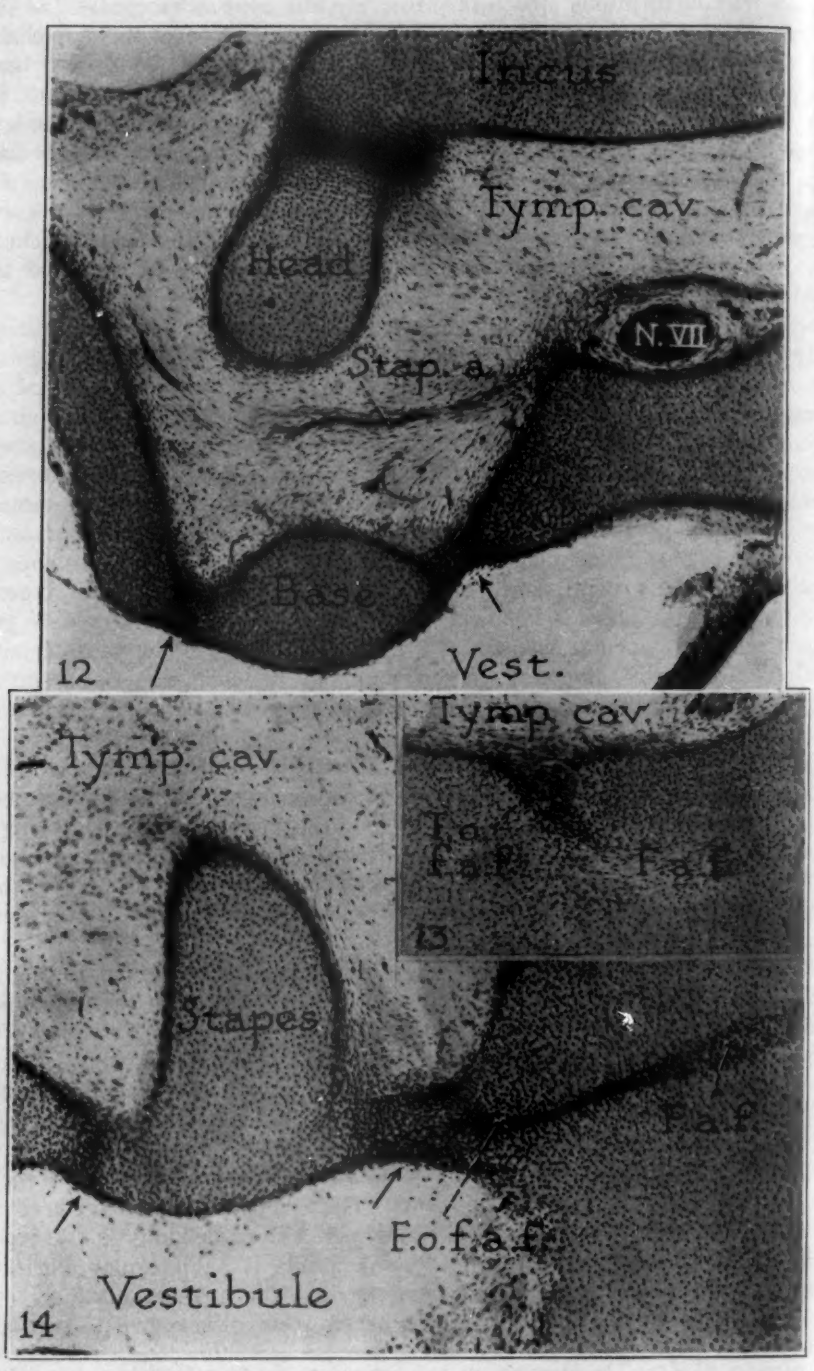
rim (fig. 15); there is no evidence at this stage of a radial orientation of fibroblasts to form a true ligamentous structure. Instead, this zone is recognized as one of active growth and histologic change. With the incorporation of the stapedial base into the lateral capsular wall, as was first observed in the 34 mm. fetus, growth changes result in histologic alterations in stapedial and capsular tissues which permit enlargement of the stapedial base in all dimensions and accommodational recession of the fenestral margin. Although cartilaginous tissue on the stapedial and that on the capsular side (fig. 17 *a*) of the primordial annular ligament (*d*) are histologically and tinctorially identical, precartilage (*c*) and early true cartilage (*b*) comprise wider zones on the stapedial than on the capsular (or fenestral) side. Growth would, therefore, appear to be far greater on the stapedial side, a difference that obtains in more advanced specimens.

The sequence of events in the process of development of the definitive stapedial and fenestral rims is as follows: Active appositional growth of the stapes base is associated with the basal perichondrium (fig. 18), while that of the basal rim is associated with the annular ligament; the annular ligament possesses chondrogenetic properties, which are demonstrated by its contributions to the stapedial rim in a sequence of cellular differentiations, represented by undifferentiated mesenchyma, precartilage, young true cartilage and mature cartilage, in succession, from the zone of growth toward the stapes (fig. 17); the cartilaginous fenestral rim undergoes concomitant dedifferentiation (from cartilage to mesenchyma) and resorption into the growth zone, which, in turn, contributes to the stapes rim. This means that the annular ligament is a zone of growth, with a definite growth increment on the stapedial side and a growth decrement on the capsular side; it maintains its identity as dense, undifferentiated mesenchymal tissue until the definitive size of the base has been attained, after which radial orientation of the cells and their processes establishes the true ligamentous primordium. While an altogether similar process of cellular differentiation and dedifferentiation characterizes the formation and development of all cavities and hiatuses in the otic capsule (Streeter,[8] 1918; Bast,[9] 1930, 1933), the feature of two opposing solid surfaces, the stapedial and the fenestral rim, receiving directly from and contributing directly to, respectively, a narrow intervening zone of undifferentiated cellular character is unique in the growth processes taking place in the development of the human skull.

The fissula ante fenestram at this stage is topographically similar to that of the 75 mm. twin fetus: An intracartilaginous fissure extends from the tympanic surface of the capsule to the vestibular surface (fig. 16), exhibiting in its course the anterior projection of altered precartilage that has characterized the fissula since its inception in the 34 mm. fetus. The tympanic orifice of the fissula is here clearly identifiable; the location of the tympanic opening is unusual, being slightly below (rather than above) the horizontal level of the anterior extremity of the stapedial rim. In downward course it is soon lost in a widened area of mesenchymal tissue representing the annular ligament. The tympanic orifice becomes immediately continuous with a fenestral cleft and with a third opening on the vestibular surface of the labyrinth. Histologically, the fissula is an irregular and often ill defined strip of precartilaginous tissue extending for a short distance into the capsular cartilage. The strip is distinguished from adjacent true cartilage by

8. Streeter, G. L.: The Histogenesis and Growth of the Otic Capsule and Its Contained Periotic Tissue Spaces in the Human Embryo, Contrib. Embryol. (no. 20) **7**:5-54, 1918.

9. Bast, T. H.: (*a*) Ossification of the Otic Capsule in Human Fetuses, Contrib. Embryol. (no. 121) **21**:53-82, 1930; (*b*) Development of the Otic Capsule: II. The Origin, Development and Significance of the Fissula Ante Fenestram and Its Relation to Otosclerotic Foci, Arch. Otolaryng. **18**:1-20 (July) 1933.



Figs. 12 to 14.—Sections through the stapedial region from a fetus of 75 mm.; × 60. Figure 13 includes the tympanic orifice of the fissula; figure 14, the fenestral orifice. Arrows point to the annular ligament.

the slight increase in number of deeply stained nuclei, relatively few vacuoles, fibrillar ramifications of the cells (characteristic of precartilage) and increased stainability of the matrix (or fibrillae) associated with the cells. At the vestibular orifice, the fissular tissue is continuous with, and indistinguishable from, the vestibular perichondrium (fig. 16).

100 Mm. Fetus (15½ Weeks).—In the 100 mm. fetus the stapedial head, like the crura and the base, has attained definitive form; the articular surface of the head is foveate; the base is flanged circumferentially, and the crura, now bowed, are clearly set off from head and base (figs. 6 and 7). The stapes has undergone decided increase in size and histologic differentiation (over the 75 and 78 mm. stages); the length of the base has been increased by one third, and the length of the stapes by one fourth, in the 25 mm. growth interval.

The vestibular window is narrow marginally, but equals in thickness the flanged base of the stapes. A distinct condensation of fibroblastic tissue between the apposed basal and fenestral surfaces marks the site of the future annular ligament.

There is striking tinctorial differentiation between the crural and the basal cartilage (figs. 19 and 20); the former is of the mature type, with deeply staining matrix; the latter, a pale-staining, young true cartilage, representing a region of active growth. A pale, peripheral growth zone of cartilage is still present, but is less prominent than the corresponding portion of the base, the capital extremity, the fenestral margin and the fissular wall. The flattened stapedial base is in intimate contact with its perichondrium; there is no longer evidence of an intermediate undifferentiated layer of cells.

Ossification has not yet begun in either the otic capsule or the stapes.

The fissula ante fenestram appears as a distinct fibrous tract in the capsular cartilage. It possesses an auxiliary (fenestral) orifice; this, like the regular orifices (tympanic and vestibular), is therefore established early.

An area of rarefaction in the capsular cartilage posterior to the inferior extremity of the vestibular window is identified as the fossula post fenestram. This structure. like the fissula ante fenestram, is produced through a histologic mechanism of dedifferentiation in cartilage that has already been formed (Bast,[10] 1938). It is usually more saccular and less regular in occurrence than the fissula.

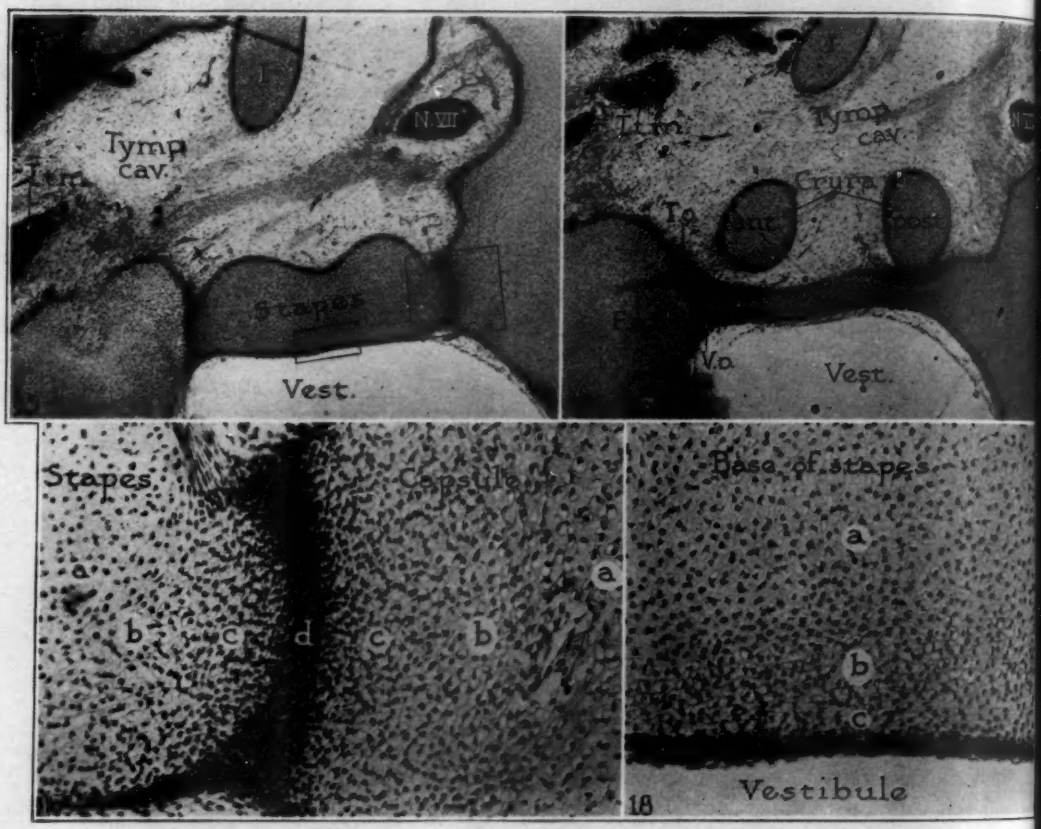
111 Mm. Fetus (16½ Weeks).—The otic capsule is still entirely cartilaginous in the 111 mm. fetus. While calcification has occurred nowhere in the capsule, there is evidence in the canalicular region of an advanced degenerative change in anticipation of this process. Bast[11] (1932) has clearly demonstrated that the outstanding modification in capsular cartilage preceding ossification is one of resorption and replacement by vascular ingrowths from the overlying perichondrium or dura mater; this process of resorption differs from that which immediately precedes ossification; the first evidence of degenerative change was observed in the canalicular cartilage of the 43 mm. stage (Bast;[11] Cauldwell and Anson,[1] 1942), and calcium deposition has not yet occurred in the 111 mm. specimen.

The stapes, likewise, is wholly cartilaginous. It has increased moderately in size since the 75 mm. stage, having added about one-third to its main linear dimensions. However, it has increased but slightly in vertical length since the 100 mm. stage and is about one tenth shorter in basal length (fig. 9; compare fig. 6). The capital extremity has a well defined foveate articular surface; the head is tilted

10. Bast, T. H.: Development of the Otic Capsule: IV. The Fossula Post Fenestram, Arch. Otolaryng. **27**:402-412 (April) 1938.

11. Bast, T. H.: Development of the Otic Capsule: I. Resorption of the Cartilage in the Canal Portion of the Otic Capsule in Human Fetuses and Its Relation to the Growth of the Semicircular Canals, Arch. Otolaryng. **16**:19-38 (July) 1932.

superiorly, meeting the crura at a slight angle—the usual relationship in the adult (osseous) stapes (Beaton and Anson,[12] 1940). The crura are bowed; the anterior crus is shorter but somewhat more massive than the posterior crus. The anterior crus arises from the extremity of the base without a definite line of demarcation between them, in contrast to the condition in the preceding stage; on the other hand, a thick basal flange clearly distinguishes the posterior crus from its area of basal origin (fig. 9). The obturator space is somewhat ovoid. The base is flattened on its vestibular surface, but gently rounded and narrowed on its tympanic aspect (fig. 9a); circumferential lipping is a prominent feature except at the anterior extremity of the base. The base tapers slightly, being wider opposite



Figs. 15 to 18.—Sections from a fetus of 78 mm. Figures 15 and 16 (× 14) represent general features; figures 17 and 18 (× 85), details of structure in areas indicated in figure 15. Here, *a* indicates mature cartilage; *b*, young true cartilage; *c*, precartilage, and *d*, dense undifferentiated chondrogenetic mesenchyma.

the posterior crus than at midbase and narrowest opposite the anterior crus. The anterior extremity of the base tends to overlap the fenestral margin, rather than to effect an accurate apposition of articular surfaces, as is constantly seen at the posterior end (figs. 26 to 28). This peculiar relationship of the stapes and the fenestral rim has already been described and figured (Anson, Karabin and Martin,[2b] 1939; figs. 34, 35, 37, 41 and 44) for postnatal stages.

12. Beaton, L. E., and Anson, B. J.: Adult Form of the Human Stapes in the Light of Its Development, Quart. Bull. Northwestern Univ. M. School **14**:258-269, 1940.

In spite of the fact that characteristic stapedial features have been acquired by the stapes of the 111 mm. fetus, the stapes of the 100 mm. stage surpasses it in clarity of delineation of the basal rim, head and neck.

The vestibular window is roughly reniform; the slight convexity of the inferior margin (fig. 8) is produced by an exaggerated upward projection of the promontory, which actually approaches the obturator foramen of the stapes (figs. 23 and 24).

Though calcification (ossification) is not in evidence, cartilaginous differentiation in the stapedial base indicates a plastic growing tissue. The mature cartilaginous crura have about them a closely applied perichondrium; the base, on the other hand, exhibits a wide cellular layer of young cartilage and precartilage in apposition with the perichondrium. This young tissue, in being added to the stapedial base, exaggerates particularly the size and shape of the low peripheral ridge of early cartilage, first observed in the 43 mm. stage. Its growth results in the formation of a definitive basal rim. The annular form of the primordial stapes is represented in the older tissue of the stapes; the outline of this deeply staining cartilage follows a curving course into the base (fig. 25). The circumferential rim, representing the rapidly growing portion of the stapes, is composed of young, pale-staining cartilage.

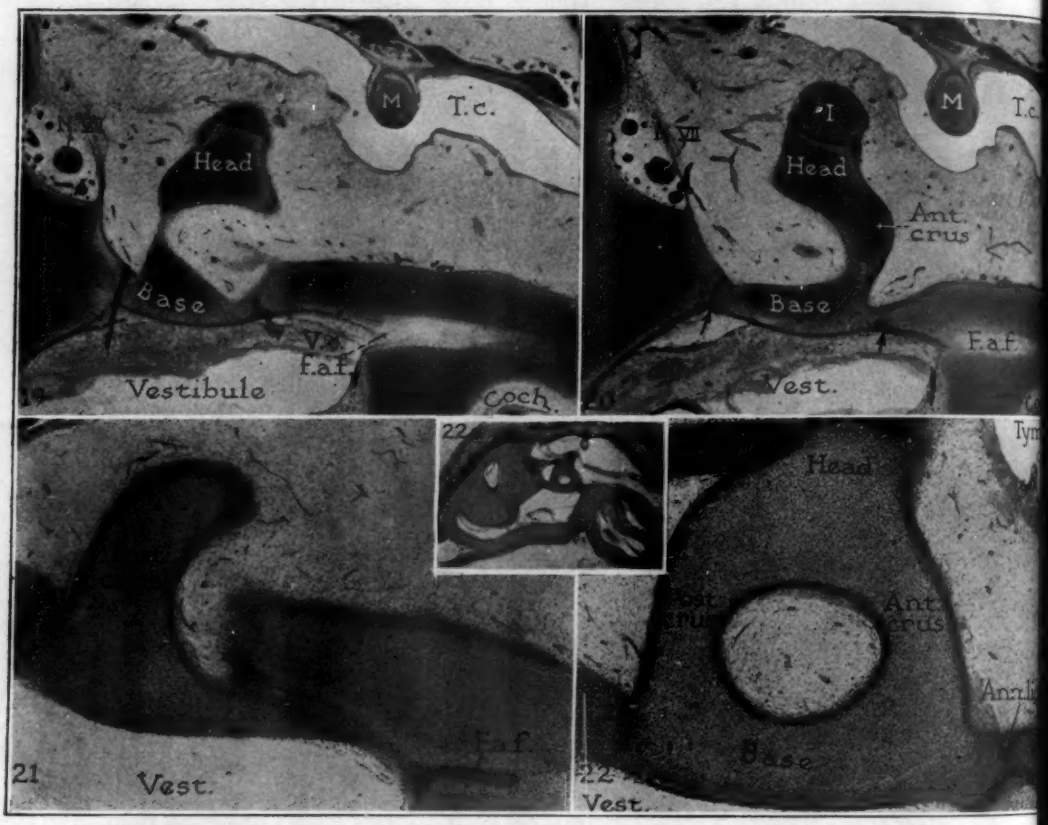
The annular ligament is a rather well defined zone of fibroblastic tissue situated at the anterior and posterior extremities and at the superior margin of the stapedial base; it has a more diffuse appearance along the inferior margin, reminiscent of earlier stages in cellular differentiation.

The fissula ante fenestram is a wide, deep groove, filled successively with necrotic cartilage, loose vacuolated mesenchyma and a delimiting plug of fibroblastic tissue. It extends from the junction of the vestibule and the scala vestibuli upward through the adjacent capsular cartilage to reach the vestibular aspect of the anterior fenestral margin (fig. 41). It then turns obliquely in a superolateral direction and passes through the fenestral rim, its constituent tissues there being continuous with the annular ligament (fig. 26); it terminates on the tympanic surface of the labyrinth as a wide, elongate, baylike orifice, immediately below the semicanal for the tensor tympani muscle (fig. 27; compare fig. 42). The core of necrotic cartilage, however, continues an intracartilaginous course above the level of the tympanic orifice (fig. 28).

The fissula is subject to many variations of form, course and cellular contents (Bast,[9b] 1933; Anson and Martin,[13] 1935). In this fetal specimen it exhibits an unusual structure, being neither a fibrous seam extending between the tympanic and the vestibular aspect of the cartilaginous capsule (78 mm. fetus) nor a cleft-like canal possessing an auxiliary orifice opening on the stapedial joint (100 mm. fetus); here it is a wide and relatively deep channel, packed with embryonic tissue and extending, without interruption, obliquely (fig. 41) across the anterior fenestral rim. As the fissula reaches the stapedial joint, the fibroblasts of the annular ligament become directly continuous and identical with those of its proximal end and with the adjacent vestibular perichondrium (figs. 25 and 26); these radially arranged cells of the ligament maintain the same direction as they continue into the fissula. Since otosclerotic change usually occurs in the region of the fissula ante fenestram, the existence of a continuous fenestral cleft, such as is present in this case, or of a more direct communication between the fissula and the stapes joint is worthy of serious consideration in the interpretation of spread of newly formed pathologic bone. Distal to the zone of fibroblastic tissue, the fissula becomes bulbous and is

13. Anson, B. J., and Martin, J.: Fissula Ante Fenestram: Its Form and Contents in Early Life, Arch. Otolaryng. **21**:303-323 (March) 1935.

occupied chiefly by an ill defined island of loose, vacuolated connective tissue, separated from the adjacent capsular cartilage by a thin layer of poorly stained fibrous tissue (fig. 26). In the anterior extremity of the fissula cartilage cells are undergoing necrotic change. When the fissula reaches the tympanic side of the labyrinth, it is invaded by numerous small blood vessels; this increased vascularization of the tympanic end persists during postnatal development (Anson and Martin,[13] 1935; Bast,[14] 1936). The tympanic orifice is 0.6 mm. long (from the fenestral rim), with a maximum width of 0.15 mm. and a minimum width (at the fenestral rim) of 0.054 mm. Its greatest depth, at the level of the vestibular edge of the fenestral rim, is 0.78 mm. The total length of the fissula, from the vestibular orifice



Figs. 19 to 22.—Sections of the stapedial region. Figures 19 and 20 (× 6), from a fetus of 100 mm.; figures 21 and 22 (× 14) and figure 22a (× 1.5), from a fetus of 112 mm. Arrows indicate annular ligament.

to the tympanic orifice, is 1.025 mm.; the perpendicular distance, between the vestibular orifice and the anterior fenestral margin, is 0.525 mm. All dimensions are greater than those given for the fissula of the 135 mm. stage (Anson, Karabin and Martin,[28] 1938).

112 Mm. Fetus (16½ Weeks).—The stapes in the 112 mm. fetus is similar in general appearance to that in the preceding stage. With high magnification

14. Bast, T. H.: Development of the Otic Capsule: III. Fetal and Infantile Changes in the Fissular Region and Their Probable Relationship to the Formation of Otosclerotic Foci, Arch. Otolaryng. **23**:509-525 (May) 1936.

certain features of stapedial growth are better demonstrated here than in the 111 mm. specimen. Thus, the base is composed of three clearly distinguishable layers (from tympanic to vestibular aspect): mature cartilage, continuous with that of the crura, consisting of small, deeply stained cells lodged in small, irregularly arranged spaces in a homogeneous chromophilic matrix; young cartilage, the cells of which lie in capacious lacunas, lineally arranged, and the matrix of which is reduced in quantity and stainability; a narrow zone of precartilage cells, and, finally, a dense perichondrium. Growth of the base through conversion of precartilage into cartilage tissue occurs, therefore, chiefly on the vestibular aspect, rather than on the obturator side. The margin of the vestibular window is, circumferentially, a young tissue, which must make adjustment for gradual enlargement of the base (see section on the 78 mm. stage).

The relation of the stapedial base to the vestibular window is a striking one: The base fits into the fenestral opening in such a way that considerable overlapping occurs along the posteroinferior margin, with the result that a portion of the cartilaginous fenestral rim projects into the obturator foramen (fig. 21). This peculiar relationship was also observed in a second 112 mm. fetus.

The fissula ante fenestram possesses an auxiliary (fenestral) orifice (fig. 22) similar to that of the 100 mm. stage, rather than a continuous vestibulotympanic cleft, as occurred in the previous (111 mm.) specimen. A fossula post fenestram was not observed.

115 Mm. Fetus (16 ½ Weeks).—There is no evidence of calcification in either the otic capsule or the stapes in the 115 mm. fetus. The stapes and the surrounding window are similar to the corresponding structures in the 112 mm. fetus.

The fissula ante fenestram closely resembles that of the 111 mm. stage; a continuous groove can be traced from the vestibule through the anterior fenestral rim and onto the tympanic surface of the labyrinth. As the fissula crosses the fenestral surface, it is so broad that the fenestral wall is strikingly deficient in this area.

120 Mm. Fetus (17½ Weeks).—Calcification of the otic capsule appears for the first time in the 120 mm. fetus. The tympanic surface of the promontory, medial to the inferior extremity of the stapes, is invaded by vascular connective tissue. This is the original site of bone formation in the fetal labyrinth.

Bast [9a] (1930) described the first center of ossification in a 126 mm. fetus as appearing "in the outer part of the capsule on the beginning of the first turn of the cochlea just anteroventral and medial to the round window." Since this center was well advanced in the 126 mm. stage, Bast concluded that it must have had its inception in a fetus between 115 and 120 mm. in length (at the age of about 17 weeks).

The stapes is still entirely cartilaginous. Its general form has not changed; the base is definitely flanged, the capital extremity being set off from the crura by a circumferential sulcus. Gradual narrowing of the vestibular window has continued, so that now the plane of its margin corresponds to that of the stapedial base.

The fissula ante fenestram appears as a definite channel in the capsular cartilage. A fossula post fenestram was not identified.

126 Mm. Fetus (18 Weeks).—Although the first center of ossification made its appearance in the temporal bone of the 120 mm. fetus, there was absolutely no indication of deposition of calcium anywhere in the cartilaginous otic capsule of the 126 mm. fetus. Bast [9a] (1930) originally described the first area of bone formation in a fetus of this length; it is apparent, therefore, that centers of ossification do not appear uniformly in specimens of comparable size or age.

The stapes is a massive cartilaginous structure (fig. 4); its dimensions exceed those of any preceding specimen and those of the ossicle in the succeeding (135 mm.) stage. The differences in relative size demonstrated by the cartilaginous stapes of the 111 and the 126 mm. fetus are entirely within the normal range of anatomic

Figs. 23 to 25.—Sections from a fetus of 111 mm.; × 33.

variation (Anson and Cauldwell,[15] 1941) and are regularly encountered in osseous specimens from adults between 25 and 75 years of age (Beaton and Anson,[12] 1940). The base is flattened and prominently lipped; the articular surface of the

15. Anson, B. J., and Cauldwell, E. W.: Growth of the Human Stapes, Quart. Bull. Northwestern Univ. M. School **15**:363-369, 1941.

head is foveate; the crura are curved and terminate laterally in a well defined neck (figs. 4, 4a, 4b, 5). The entire stapes is invested by a heavy perichondrium, the thickness of which, together with the presence of a peripheral zone of young cartilage, indicates rapid appositional growth. The tympanic mesenchyme, in loose cellular continuity with the perichondrium, is highly vascularized (fig. 31). The vestibular window is like that of the preceding stage; its margins are separated from the apposing basal rim of the stapes by a definite, dense annular ligament (fig. 30).

The fissula in this specimen possesses a remarkable form; it is not chiefly intracapsular, with limited vestibular and tympanic communications, but opens on the extracapsular spaces as an uninterrupted, deep sulcus. When viewed from the medial aspect, it appears as an oblique cleft sent downward and forward from the anterior margin of the vestibular window (fig. 32); when the structure is seen from the fenestral aspect, its opening is observed to broaden as it descends to the beginning of the scala vestibuli (fig. 33). After crossing the window, it ascends on the tympanic wall, this lateral part of its opening moving away from the window and toward the cochlea in its ascent (fig. 34). When the fissula is treated as content rather than as excavation—as a "cast," in other words—the feature of continuity is even better portrayed; the fissula follows a course which, without interruption, makes it an invagination of, successively, tympanic, fenestral and vestibular connective tissue (fig. 35). This is the fifth specimen in the current set in which a fissula of the continuously open type occurred (see 75, 78, 111 and 115 mm. series). In the three older specimens the opening of the fissula follows an oblique and somewhat sinuous course, approaching a horizontal plane more closely in the intermediate, or fenestral, portion than in the vestibular, or tympanic, portion (compare figs. 33 and 41; figs. 34 and 42). But in the 126 mm. fetus the fissula is much longer than in the other two, and its extremities are farther removed from the vestibular window.

At this stage of development, dedifferentation of both cartilage and connective tissue can be observed at the anterior (distal) extremity of the fissula (figs. 29 and 30). This indicates that the fissular space is still undergoing enlargement, even after its margins have been formed in true cartilage. The observation leads to the conclusion that there can be no regularity in the process of fissular growth; some fissulae remain small, while others become widened and lengthened by secondary erosion of their cartilaginous walls.

135 Mm. Fetus (19 Weeks).—In the 135 mm. fetus the structure of the stapes and the otic capsule shows little advance over the condition of these parts in the preceding stage (Anson, Karabin and Martin,[2a] 1938, figs. 24 to 27).

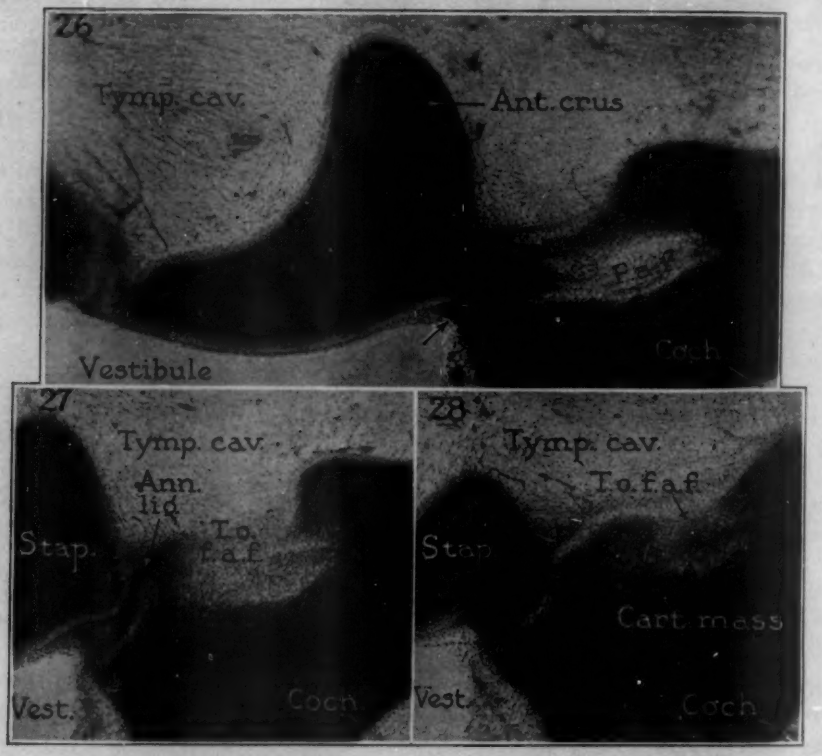
147 Mm. Fetus (20 Weeks).—Whereas ossification of the medial and lateral aspects of the vestibule and cochlea has advanced beyond that of any other specimen up to this stage of development, the otic capsule in the region of the vestibular window and fissula is still cartilaginous.

The stapes is similar to that of the 135 mm. stage; it is entirely cartilaginous and offers no indication of approaching calcification.

The fissula ante fenestram has capacious tympanic and vestibular orifices and a small, slitlike auxiliary (fenestral) orifice.

A fossula post fenestram with a wide vestibular orifice is located posterior to the oval window. This topographic feature places the fossula in group I in the system of classification suggested by Bast,[10] since it communicates with the vestibular cavity only (73.3 per cent of fossulae); it is also of type I, because it is widely open to the vestibule (23.3 per cent of group I).

150 Mm. Fetus (20 Weeks).—Ossification of the otic capsule began in the 120 mm. fetus. At the 150 mm. stage all but one of the fourteen centers of ossification have appeared (Bast, [9a] 1930); however, fusion of two or more centers occurs so rapidly after their inception that the capsule has the appearance of being invaded by large, irregular, patchy masses of bone. Fusion of originally separate ossification centers results in the production of five major areas of bone: (1) the labyrinthine wall at the base of the promontory, extending superiorly and medially from the region of the fenestra cochleae and approaching the inferior margin of the rim of the oval window; (2) a wide strip along the medial cochlear wall, spreading to the anterior surface of the internal auditory meatus; (3) a roughly triangular area involving the basal portion of the modiolus, wherefrom it projects into the ill defined



Figs. 26 to 28.—Sections from a fetus of 111 mm.; × 34. Figure 26, fenestral orifice of the fissula; figures 27 and 28, tympanic orifice.

saccular recess, completely surrounds the superior cribriform aperture with a bony collar and extends anteriorly onto the posteromedial surface of the spiral septum; (4) the facial sulcus and the adjacent canalicular area opposite the superior margin of the stapes and, to a lesser extent, the corresponding vestibular region, and (5) an area of calcified cartilage located on the medial margin of the superior semicircular canal.

It is important to record that the cartilaginous tissue of the general fenestral and fissular areas undergoes ossification more slowly than that forming the remainder of the capsule. This is to be expected, since, as was shown in an earlier article, the cartilage persists throughout life as the wall of the vestibular window and of the vestibular extremity of the fissula (Anson, Karabin and Martin,[2a] 1938, figs. 20 to 23).

An advanced degree of ossification is exhibited on the wall of the promontory. Here both the tympanic and the cochlear surface consist of deep-staining plates of periosteal bone; these plates are fenestrated at intervals to permit passage of osteogenic buds from the periosteal surface into the excavated interior. Foramens are smaller and fewer on the vestibular surface. The intervening spaces are heavily vascularized, and interstices between blood vessels are filled with primitive marrow cells. Irregularly shaped islands of intrachondral bone are distributed throughout this vascular and cellular matrix. Numerous osteoclasts maintain intimate contact with these osseous masses. In older fetuses identical processes are observed in the stapes.

The stapes has now acquired relatively massive proportions (fig. 10): Its vertical length is eight times and its basal length ten times that of the corresponding dimensions of the stapes in the 25 mm. embryo; both measurements are three and one-half times as great as in the 50 mm. stage. The head is deeply foveate, for articulation with the lentiform process of the incus, and has a slight downward (caudal) inclination. A definite neck, formed by the junction of widely divergent crura, can be distinguished.[16] The crura are long, the anterior crus being the shorter, straighter and less massive of the two; the posterior crus possesses a rather pronounced curve where it joins the neck. The intercrural (obturator) foramen is a relatively small oval hiatus; its long axis coincides with that of the stapes base. The small size of the obturator space is due to the unusual thickness of the base (fig. 10 a).

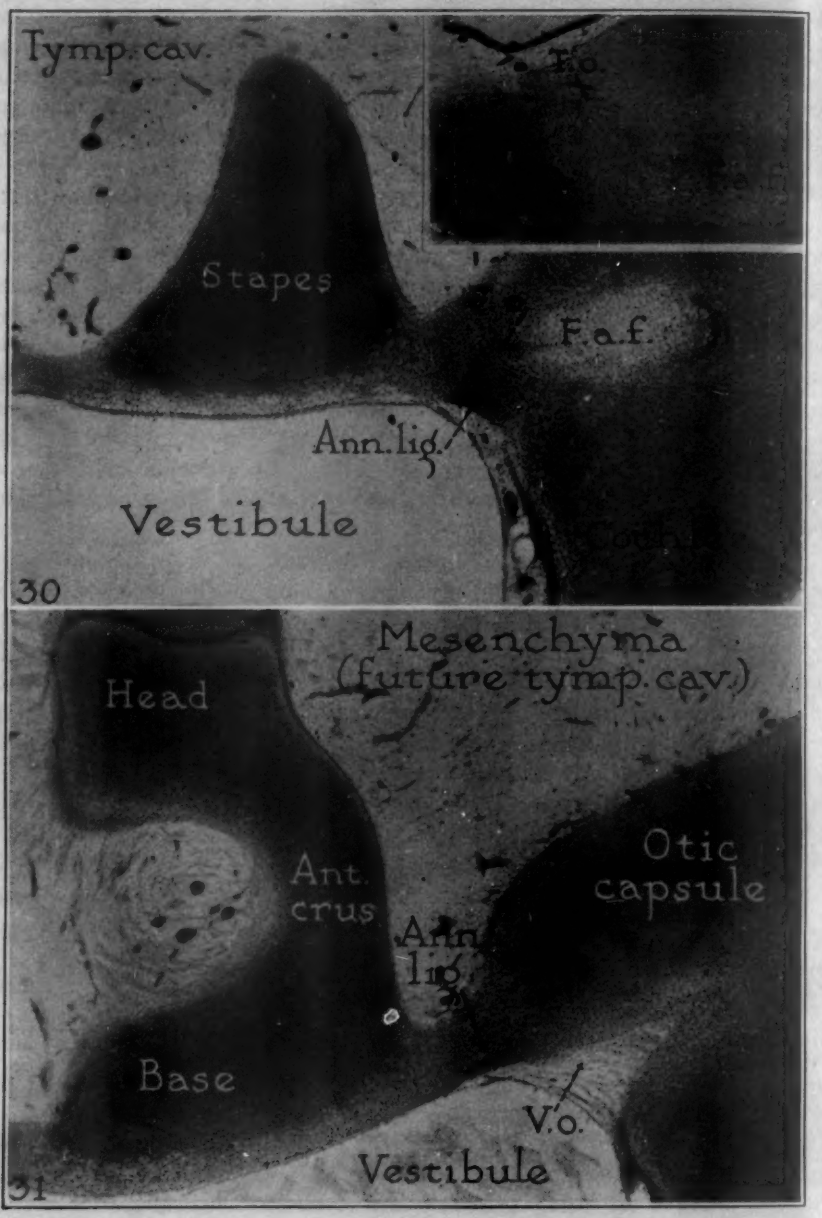
A prominent basal lip has developed; it is thick and tapering anteriorly and thick and flattened posteriorly; it is somewhat thinner on the superior and inferior than on the anterior and posterior margins. From the vestibular aspect, the base is elongate and slightly reniform; its anterior extremity tapers sharply, while the posterior end is well rounded (fig. 11). A prominent convexity is present on the vestibular surface of the base, opposite the obturator aspect of the anterior crus (fig. 10). An umbo of this sort is not a feature of the adult human stapes, but regularly occurs on the stapedial foot plate in some of the Simidae (Hylobates), the Cebidae and in Castor and Hyrax (Doran,[17] 1878). The tympanic portion of the base, as previously mentioned, is unusually thickened and sharply rounded on its obturator surface (fig. 10 a).[18] An irregular, but well defined, center of ossification appears on this surface and extends outward on the posteroinferior aspect of the

16. In the 25 mm. embryo the basal portion of the cartilaginous stapes is distinguishable through slight flattening; a capital projection, however, has not appeared (fig. 1). In the 40 mm. embryo a capital end is identifiable only in respect to its apical position (Anson, Karabin and Martin; [2] fig. 6); in the stapes of the 50 mm. fetus a true projection is evident, extending beyond the outline of the original ring (fig. 2). This capital eminence increases in all dimensions as development proceeds; in the 100 mm. stage it is foveate and slightly lipped (fig. 3); in the stapes of the 126 mm. (fig. 4) and the 150 mm. fetus (fig. 10) the features of foveation and lipping are even more pronounced. As soon as the articular portion of the medial extremity becomes thus broadened and circumferentially lipped, it may be said—for purposes of description—that both the stapedial head and the neck are distinguishable; the head is then primarily the articular part, while the neck is the constricted area between the latter and the point of origin of the crura. In adult specimens these subdivisions are usually definable; however, in some postnatal stapes head and neck merge indistinguishably.

17. Doran, A. H. G.: Morphology of the Mammalian Ossicula Auditus, Tr. Linn. Soc. London (Ser. 2, zool.) **1**:371-496, 1878.

18. It is J. Hegetschweiler's opinion (Die embryologische Entwickelung des Steigbügels, Arch. f. Anat. u. Entwcklngsgesch., 1898, pp. 37-56) that incomplete resorption of the ridgelike obturator surface of the base is responsible for the occasional appearance in the adult stapes of a crista stapedis, a bony ridge traversing the long axis of the tympanic aspect of the foot plate.

posterior crus (fig. 10).[19] Dreyfuss,[20] (1893) observed the earliest area of ossification of the base and crura in a fetus of 19 to 20 weeks, whereas Broman,[21] (1899) stated that the first center made its appearance in the region of the base in a

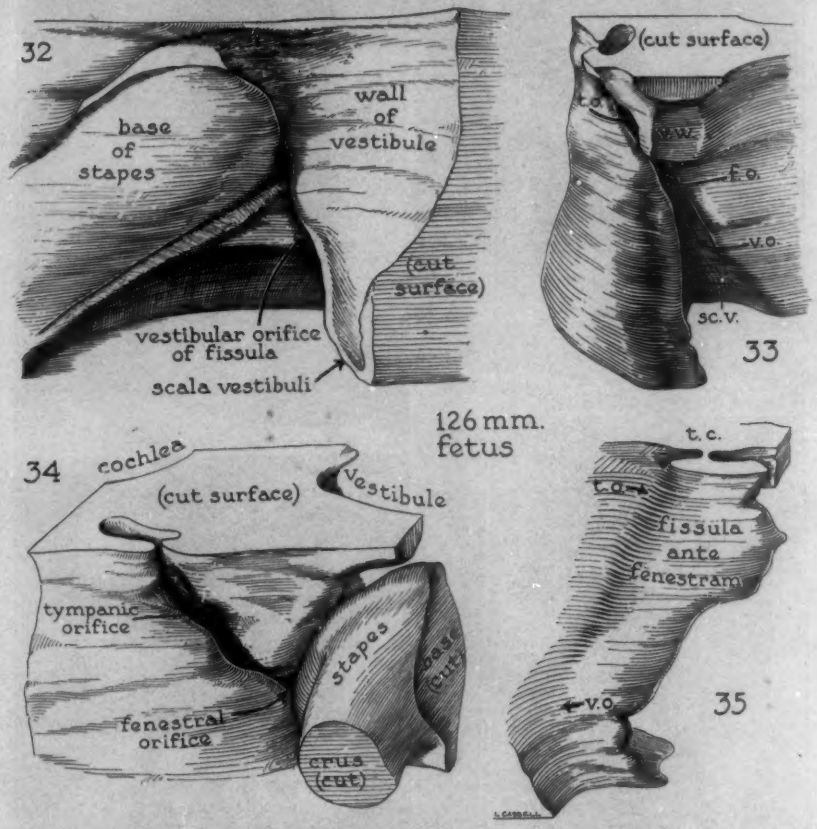


Figs. 29 to 31.—Sections from a fetus of 126 mm.; × 34. Figure 29, tympanic orifice of the fissula; figure 30, fenestral orifice; figure 31, vestibular orifice.

19. As pointed out in an earlier article (Anson, Karabin and Martin[2]), a center of ossification was present on the basal portion of the stapes in the 161 mm. fetus; it is an area of calcification in which true bone has not yet appeared. It should be recorded that in the article referred to

fetus of 210 mm. total length (about 142 mm. crown-rump length, according to Noback's formulas). Oesterle[22] (1931), likewise, described primary basal ossification in the stapes of a 4 to 5 month (16 to 20 week) fetus. Unfortunately, neither Dreyfuss nor Oesterle indicated the measured length of his specimen.

Although advanced features of ossification are well demonstrated on the promontory, the entire sequence of events, from the earliest histologic modification of the cartilage cells to the formation of periosteal bone, is revealed in the serial sections of the stapes. That structure is composed of mature cartilage, except at the periphery of its basal rim and vestibular surface; in these regions a narrow layer of flattened, concentrically arranged cells is identified, an indication of con-



Figs. 32 to 35.—Reconstructions of the stapedial and fissular regions in a fetus of 126 mm.; × 17. Figure 32 is a vestibular (medial) view; figure 33, an anterior fenestral view; figure 34, a tympanic (lateral) view, and figure 35, a tympanic view of a fissular cast.

tinued cartilaginous growth. Ossification of the stapes begins at the periphery (fig. 38) and gradually invades the base and the posterior crus (figs. 36 and 37). The first noticeable change in the cartilage cells preparatory to formation of bone

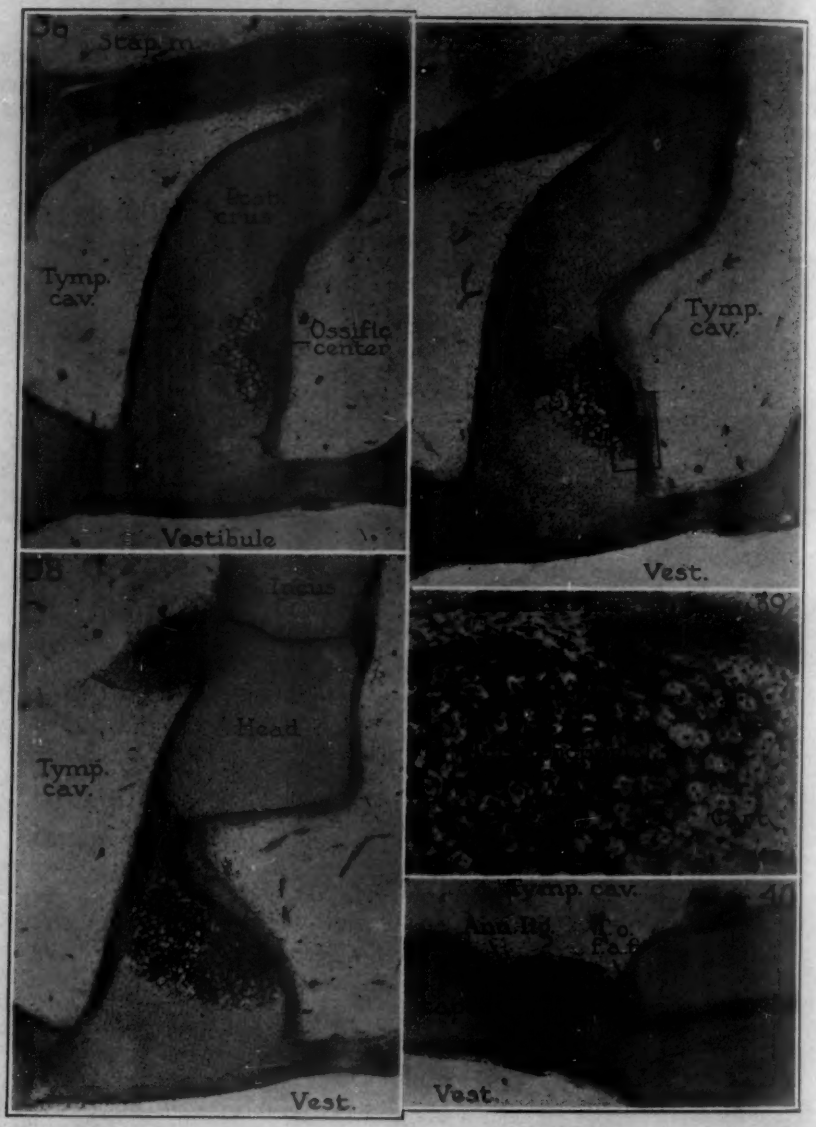
figure 30 is a photomicrograph from the 161 mm. series (erroneously identified as of the 183 mm. fetus).

20. Dreyfuss, R.: Beiträge zur Entwicklungsgeschichte des Mittelohres und des Trommelfells des Menschen und der Säugethiere, Morphol. Arb. **2**:607-662, 1893.

21. Broman, I.: Die Entwicklungsgeschichte der Gehörknöchelchen beim Menschen, Anat. Hefte **11**:509-670, 1899.

22. Oesterle, F.: Ueber den Feinbau der Gehörknöchelchen und seine Entstehung, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:311-327, 1933.

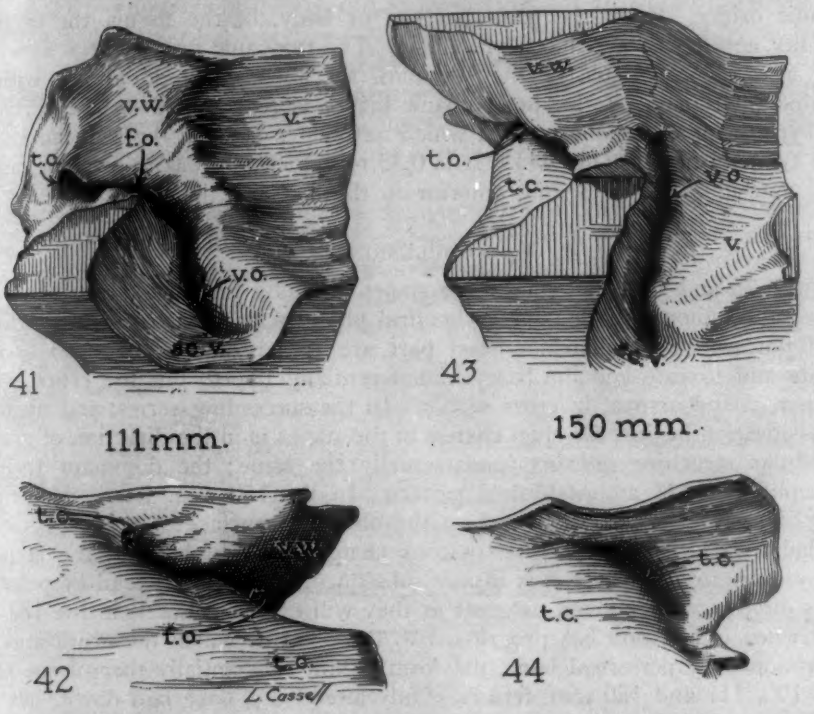
is pronounced swelling of the lacunas; the cytoplasm shrinks from the walls, and nuclei become pyknotic and irregular. This zone occupies the periphery of the calcifying area. Superficial to these cells the cartilage matrix is more deeply stained and has a trabeculate appearance; its increased affinity for hematoxylin is indicative of deposition of calcium. The overlying perichondrium is ruptured



Figs. 36 to 40.—Sections from a fetus of 150 mm.; × 24. Figure 39 shows a detail of the area indicated in figure 37.

by the vascular connective tissue buds which penetrate the cartilage mass; the connective tissue cells (osteoblasts) invade the swollen lacunas and replace the cartilage cells (fig. 39). The appearance of blood vessels within the cartilage presages vigorous excavation of that tissue. But this process is as incomplete as it is rapid, as evidenced by the areas of cartilage left in the wake of the advancing

vascular buds. Periosteal bone has appeared over the zone of calcified cartilage and takes the form of an incomplete, perforated crust directly continuous with the adjacent perichondrium. The bony plate is actually covered by a layer of mesenchymal tissue, thicker but less compact than inactive perichondrium. The entire layer consists of two zones: (1) an outer stratum of longitudinally directed mesenchymal cells, and (2) an inner stratum of larger, spindle-shaped cells. The inner stratum is in intimate contact with the bony plate where the latter is unbroken and invasive where foramens allow passage into the excavated cartilage. These cells become osteoblasts associated with the formation of periosteal and endochondral bone.[23] The changes observed in the ossification of the stapes are seen to be precisely those operating in the labyrinth.



Figs. 41 to 44.—Reconstructions of the fissular region in fetuses; × 18. Figures 41 and 42, a fetus of 111 mm.; figures 43 and 44, a fetus of 150 mm.

Except for minor topographic differences, the fissula ante fenestram of the 150 mm. fetus more closely resembles that of the 50 mm. stage than any other in the present group of fetal series. Throughout its course in the capsular cartilage the fissula is a narrow, elongate seam, composed of dense connective tissue. The vestibular orifice of the fissula is a low, narrow opening in the cartilaginous wall of the vestibule and scala vestibuli (fig. 43); into it extends the vascular, fibroblastic tissue of the vestibular perichondrium. The form of the orifice in this and in other growing stages is, of course, dependent in part on the degree of resorption of the chondral wall and on the extent to which the connective tissue is converted into cartilage; during enlargement of the capsule its vestibular termina-

23. These cartilage islands are eventually invaded and their cells replaced by those of osteogenetic buds, with the resulting formation of intrachondral bone. When a mass of intrachondral bone is well organized, endochondral bone is deposited on and about it.

tion must retreat and its distal, or cochlear, extremity become progressively farther removed from the site of primordial formation (Bast,[11] 1932).

The vestibular orifice of the fissula has an average width of 0.1 mm. and a perpendicular height of 0.84 mm.; the length of the orifice, measured along the curving wall of the vestibule and scala, is 1.63 mm. As the fissula extends inward its main mass is somewhat offset from the vestibular portion—a morphologic feature which suggests resemblance to an alar projection. The projection extends inferiorly on the lateral aspect of the vestibular portion, so that in sections it appears as a separate, but parallel, bay of connective tissue. Anteriorly the main portion of the fissula extends to within a short distance of the cochlea; posteriorly it almost reaches the vestibular window, just failing to establish an auxiliary opening on the annular ligament; superiorly it rises well above the level of the tympanic orifice. From the main portion, or body, of the fissula the tympanic extremity appears as a reduced projection. The tympanic orifice turns at a right angle, as does the fissula of the 126 mm. specimen, to communicate with the tympanic cavity (fig. 40). The tympanic orifice is represented on the surface of the labyrinth by an oblique groove, which terminates beneath the semicanal of the tensor tympani muscle (fig. 44). It is 0.35 mm. long and 0.05 mm. wide, and its caudal extremity lies 0.33 mm. anterior to the stapedial joint.

CONCLUSIONS

In the 75 and 100 mm. stages, the stapes attains a form which, in general, is retained throughout the remaining chondral phase of its development; a cylindric capital portion and a platelike basal part are recognizable. The head becomes elongate and foveate and the base circumferentially lipped, but the crura remain columnar, round or oval in cross section. In the succeeding series, and up to the time ossification begins, the chief change in the stapes is in the direction of growth; the cellular structure remains fundamentally the same; the dominant theme is the amplification of an established pattern. In the 150 mm. fetus, the primary center of ossification has appeared on the obturator surface of the base. It is a remarkable fact that at the time osseous change is first observed the stapes is actually more massive than it is in any subsequent stage, either fetal or postnatal, and its dimensions are about as great as they will ever be. Later, in the 183 mm. fetus,[3] when ossification has progressed to the point at which the crura and base are enveloped by periosteal bone, the form is still substantially that of the stapes of the 100, 111 and 150 mm. fetuses. Only after bone, once laid down, has been partially removed does the stapes assume the channeled form which characterizes the "adult" ossicle.

The crura are first set off from the base and head in the 34 mm. fetus. Crural growth is appreciable in succeeding series; the columns not only lengthen but become separately distiguishable parts through their relation to the broadening base: They seem to be implanted in a plate the margins of which extend beyond the areas where the crura and base become continuous. With onset of ossification in the stapedial base, the posterior crus is invariably the first to be involved.

In the 100 and 111 mm. stages the capital extremity is cylindric, deeply foveate on its lateral (incudal) surface and widely mergent with medial crural extensions; an ill defined neck now makes its appearance, both the head and the neck approaching their definitive external shape, although they are continuous parts of a solid, cartilaginous cylinder.

As soon as the process of ossification spreads to the crura and neck, growth of the stapes ceases; thereby, in the 183 mm. fetus, is produced a continuous tube of bone of modified annular form, cartilage remaining as a basal plate and a

capital cylinder. Once formed in bone, subsequent changes are of a unique order: All periosteal bone facing the obturator foramen is destroyed, and together with it the transitory endochondral bone and associated marrow which originally were contained within the neck, crura and head. Through the gradual spread of tympanic mucous membrane, the submucosal tissue comes to occupy these excavated portions of the ossicle.

The tissue of the annular ligament does not differentiate with equal rapidity over its several portions; its development seems to be consistently retarded on the inferior stapedial margin; on other margins it consists of dense, radially directed fibroblastic tissue, in all stages beyond that of 126 mm.

The vestibular (oval) window is a well defined hiatus conforming closely to the outline of the stapedial base (foot plate) in the 75 and 100 mm. fetuses. The shape of the window is apparently altered by the upward growth of the promontory (111 mm.), by stapedial overlapping of the fenestral margin (112 mm.) or by marginal overlapping of the stapedial base (anterior extremity of base, 150 mm.). The window approaches its adult dimensions in the 150 mm. fetus; changes toward the adult form are practically complete as early as the 183 mm. stage. The fenestral margin is never completely ossified; the articular edge remains a rim of cartilage throughout life.

The fissula ante fenestram makes its appearance in the 34 mm. fetus; in later stages, exclusive of the 150 mm. stage, a series of regressive and progressive changes in its constituent tissues mark this area as one of histologic instability. A tympanic orifice is vaguely suggested in the 50 mm. fetus, but is well established in the 75 mm. fetus and in succeeding stages. Variations in fissular shape and extent are multifold: An intracartilaginous seam may extend from the tympanic to the vestibular surface; an auxiliary, or fenestral, orifice may appear; a deep wide cleft may pass from the vestibule, through the anterior fenestral margin and onto the tympanic surface of the labyrinth. In every specimen studied the tympanic orifice of the fissula is located more superiorly than the vestibular; the latter is regularly the larger of the two and the closer to the fenestral margin.

The fossula post fenestram is an inconstant channel in the capsular wall situated posterior to the vestibular window, as its name implies; it is present in the 100 and 147 mm. fetuses and in both communicates only with the vestibular cavity.

303 East Chicago Avenue.

CRITERIA FOR THE SELECTION OF TREATMENT OF CANCER OF THE LARYNX

CHEVALIER L. JACKSON, M.D.
AND
JOHN V. BLADY, M.D.
PHILADELPHIA

Much has been written about the relative advantages of irradiation and surgery in the treatment of cancer of the larynx, and too often these two methods have been considered from a competitive standpoint. Martin,[1] of Memorial Hospital, New York, in his paper read before the national meeting of the American Laryngological, Rhinological and Otological Society in June 1940, pointed out what he called the "futility of the partisan concept." A similar broad, impartial viewpoint was taken by Hautant[2] in his excellent discussion of this subject presented at the Third International Congress of Otorhinolaryngology in Berlin in 1936.

The methods of treatment of cancer of the larynx which we accept as appropriate are laryngofissure, laryngectomy and irradiation by roentgen rays.[3]

Laryngofissure[4] is performed by the "clipping" operation if the lesion does not reach the anterior commissure and by the Jackson[5] "anterior commissure" technic if it does reach the commissure. The technic of laryngectomy also varies somewhat with the character of the individual lesion. For example, in cases in which there is thought to be no possibility of involvement of cartilage the subperichondrial operation advised

---

Read before the Southern Section of the American Laryngological, Rhinological and Otological Society, Inc., Atlanta, Ga., Jan. 23, 1942.

1. Martin, H.: Selection of Treatment for Cancer of the Larynx, Ann. Otol., Rhin. & Laryng. **49**:728 (Sept.) 1940.

2. Hautant, A.: La radiothérapie des cancers intra-laryngés. Ses indications et celles de la chirurgie, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:15, 1936.

3. (*a*) Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939. (*b*) Jackson, C. L.: Treatment of Cancer of the Larynx, South. M. J. **34**:243 (March) 1941. (*c*) Blady, J. V., and Chamberlain, W. E.: Treatment of Cancer of the Larynx by Roentgen Rays (Report of Five Year End Results), Am. J. Roentgenol., to be published.

4. Jackson, C. L.: Laryngofissure for Cancer of the Larynx: Observations Based on a Series of Fifty Consecutive Cases, Arch. Otolaryng. **33**:520 (April) 1941.

5. Jackson, C.: The Results of Operative Methods in the Treatment of Cancer of the Larynx, Symposium, Proc. X^{e} Cong. internat. d'otol., Paris, 1922; Ann. d. mal. de l'oreille, du larynx **41**:1221, 1922.

by Crowe and Broyles [6] may be done. If the lesion is located on the ventricular bands or at the base of the epiglottis, the technic advised by Bisi [7] of Buenos Aires (removal of the entire epiglottis, preepiglottic space and hyoid bone) is preferred. If the lesion is strictly cordal or subglottic, the epiglottis may be amputated at its base and the greater portion left in situ. If the lesion does not extend subglottically a large section of the cricoid cartilage may be saved and a better stoma thus obtained. Subglottic extension may require the removal of not only the whole cricoid cartilage but several tracheal rings as well.

The technic of treatment by roentgen rays that we have used is fully described in a recent paper by one of us (J. V. B.) and Chamberlain.[3c] Cancer of the larynx without cervical metastases is treated exclusively with roentgen rays, whereas cancer of the larynx with metastases to the nodes of the neck is treated by external irradiation with roentgen rays, followed by surgical exposure, excision whenever the metastases are confined within their capsule and in either case implantation of gold radon seeds. Interstitial irradiation with radon or radium for treatment of a primary growth is not employed in our clinic.

SELECTION OF TREATMENT

The major considerations in the selection of a method of treatment are, first, the prospect of cure of a fatal disease and, second, conservation of function, in other words, voice. Among the secondary considerations are duration of morbidity, degree of discomfort and possibility of complications.

In our opinion there is little difficulty in the choice between laryngofissure and laryngectomy. Neither has there been difficulty in the choice between laryngofissure and irradiation thus far, although recent reports on the roentgenologic treatment of early intrinsic cordal tumors do suggest hopeful results. In our clinic, as well as in many others, however, laryngofissure is advised in preference to irradiation because of its long-established record of excellent end results. The discomfort of the patient after laryngofissure is much less than after irradiation; on the other hand, though the patient with laryngofissure will have a good useful voice in most instances, the voice after irradiation is usually even better.[3c, 8]

When it comes to the choice between laryngectomy and irradiation the decision is more difficult. If definite criteria for selection of treat-

6. Crowe, S. J., and Broyles, E. N.: Carcinoma of the Larynx and Total Laryngectomy, Ann. Otol., Rhin. & Laryng. **47**:875 (Dec.) 1938.

7. Bisi, R. H.: Cancer laringeo, Buenos Aires, Libreria y Editorial "El Ateneo," 1938.

8. Jackson, C. L.: The Voice After Laryngofissure and Laryngectomy for Cancer of the Larynx, Surg., Gynec. & Obst. **70**:537 (Feb. 15) 1940.

ment by irradiation could be established, which would offer as good a prospect of cure as laryngectomy, of course irradiation would be preferable. This is said despite the fact that we have seen abundant proof that the laryngectomized patient almost always quickly adjusts to his handicap, and very often is able to resume his original occupation. The laryngologist must not be satisfied with the status of his knowledge as long as in many cases he is simply inclined to say, "This patient could be

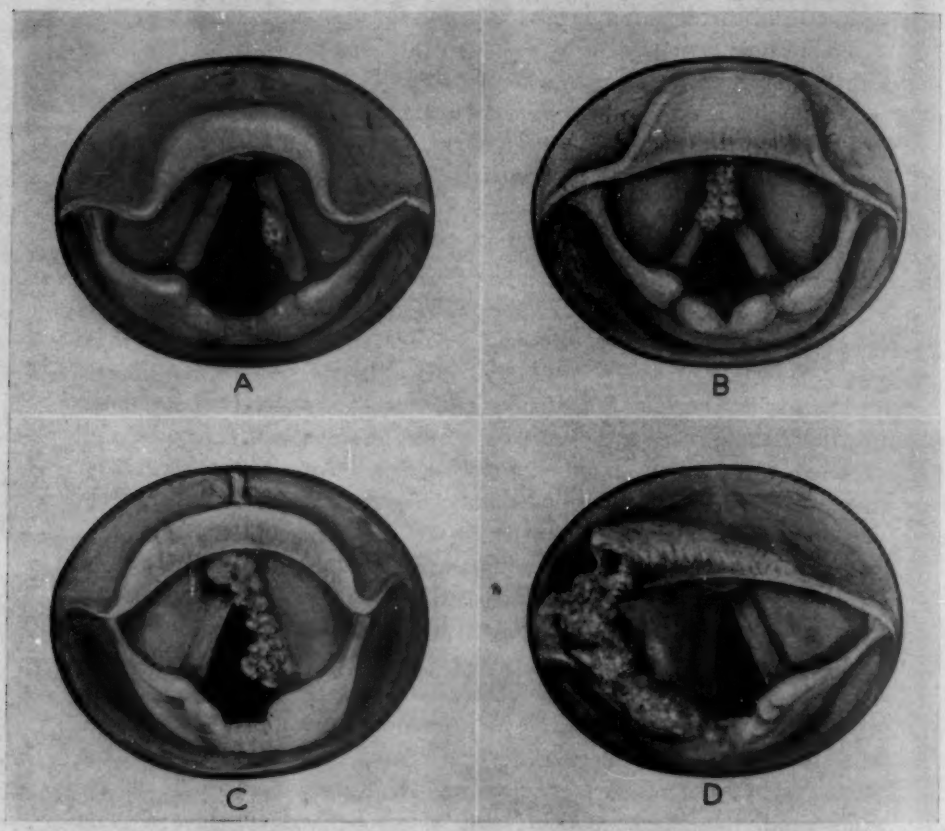


Fig. 1.—Lesions *A* and *B* are both amenable to extirpation by laryngofissure; lesion *C* would require laryngectomy or irradiation, and lesion *D* should be treated only by irradiation (from Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939).

treated either way!" He must continue to collect, review and analyze his material in the effort to improve his knowledge and judgment on this point.

Criteria.—Our criteria for the selection of a method of treatment of cancer of the larynx are: (1) the presence or absence of cervical

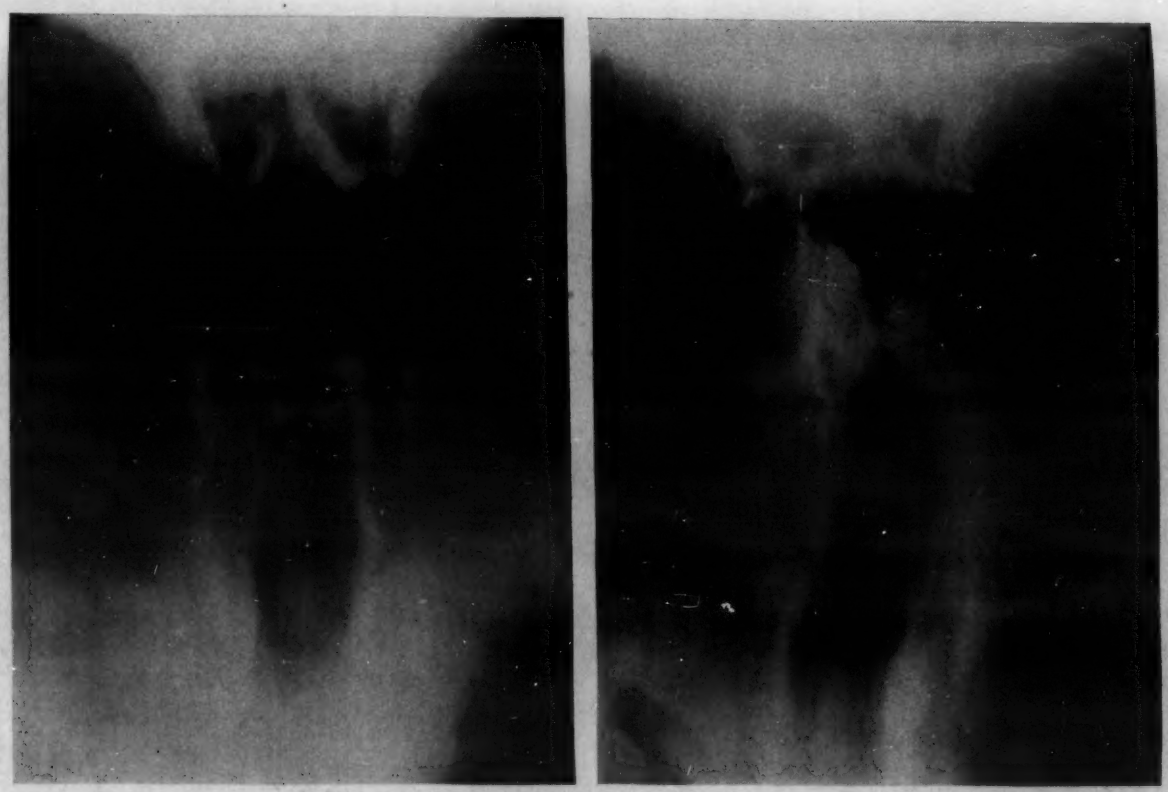


Fig. 2.—Planigraphy is of considerable value as an aid in demonstrating the location and extent of tumors of the larynx. The view at the left shows the appearance of the normal larynx; the view at the right, a carcinoma of the right side obscuring the ventricle and encroaching on the pyriform sinus. The left ventricle is obscured by edema. Planigraphy is of especial value in demonstrating subglottic extension (from Jackson, C., and Jackson, C. L.: Cancer of the Larynx, Philadelphia, W. B. Saunders Company, 1939).

metastases; (2) the location and extent of the lesion, as evidenced by its appearance on direct and indirect laryngoscopy (fig. 1) and by lateral and planigraphic (fig. 2) roentgenographic study; (3) the motility and mobility of the laryngeal structures; (4) the histologic character of the lesion, and (5) the general physical condition and temperament of the patient.

Indications for Laryngofissure and Laryngectomy.—1. Lesions occupying the middle third of one vocal cord are suitable for laryngofissure by the "clipping" technic (fig. 3).

2. Lesions reaching the anterior commissure and even involving the opposite cord are also amenable to extirpation by the laryngofissure route, but in such cases the "anterior commissure" technic [5] should be used.

3. Lesions in which the growth is cordal but has reached the posterior end of the cord and produced impairment of motility, or has

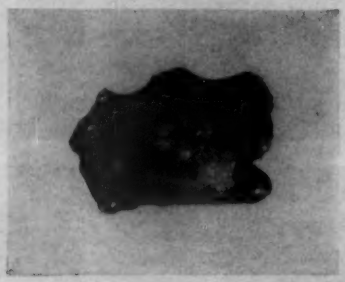


Fig. 3.—Ideal type of lesion for extirpation by laryngofissure. This operative specimen represents one vocal cord, with the lesion occupying its middle third.

extended subglottically, ordinarily call for total laryngectomy. To quote St. Clair Thomson,[9] impaired mobility is "of evil augury" (fig. 4).

4. Lesions in which the tumor has invaded cartilage (but not muscles) also call for laryngectomy, provided there are no metastases.

Indications for Irradiation.—1. Lesions unsuitable for laryngofissure and for which laryngectomy is contraindicated by the age, physical condition or temperament of the patient (to quote St. Clair Thomson again, "age, health or prejudice"). These factors rarely, if ever, contraindicate the less radical procedure of laryngofissure, but they are to be regarded as relative contraindications to laryngectomy.

2. "Inoperable" growths which are extrinsic by origin or extension or which have cervical metastases. The latter must be treated by

9. Thomson, St. C.: Intrinsic Cancer of the Larynx: The Occurrence of Impaired Mobility of the Affected Cord and Its Value in Diagnosis and Prognosis. Lancet **2**:220, 1928.

protracted irradiation, followed immediately by implantation of radon, either through the skin or after surgical exposure (fig. 5).

3. Lesions which have reached the posterior extremity of the cord but have not produced impairment of motility of the cord. Such growths constitute a group for which laryngofissure is contraindicated but for which irradiation may be preferred to laryngectomy. It is the consensus at present that impairment of motility contraindicates laryngofissure, and while such impairment is not a contraindication to irradiation, it

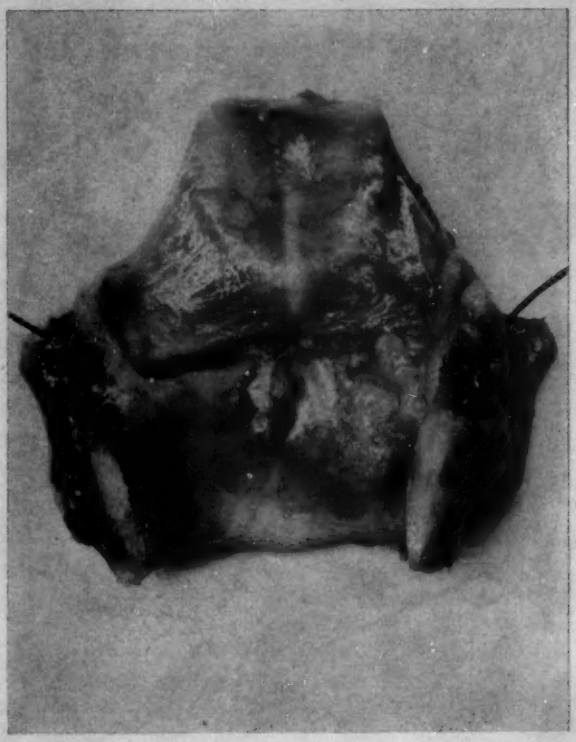


Fig. 4.—This type of lesion is best treated by total laryngectomy. The subglottic extension and cordal fixation contraindicate an attempt at extirpation by laryngofissure, and these characteristics also render the lesion unsuitable for treatment by irradiation.

is not a favorable sign; complete fixation of one or both sides of the larynx, on the other hand, is considered even less favorable.

HISTOPATHOLOGY

There are those who minimize the importance of the histopathologic picture, but we believe that we are safe in saying that the ultimate

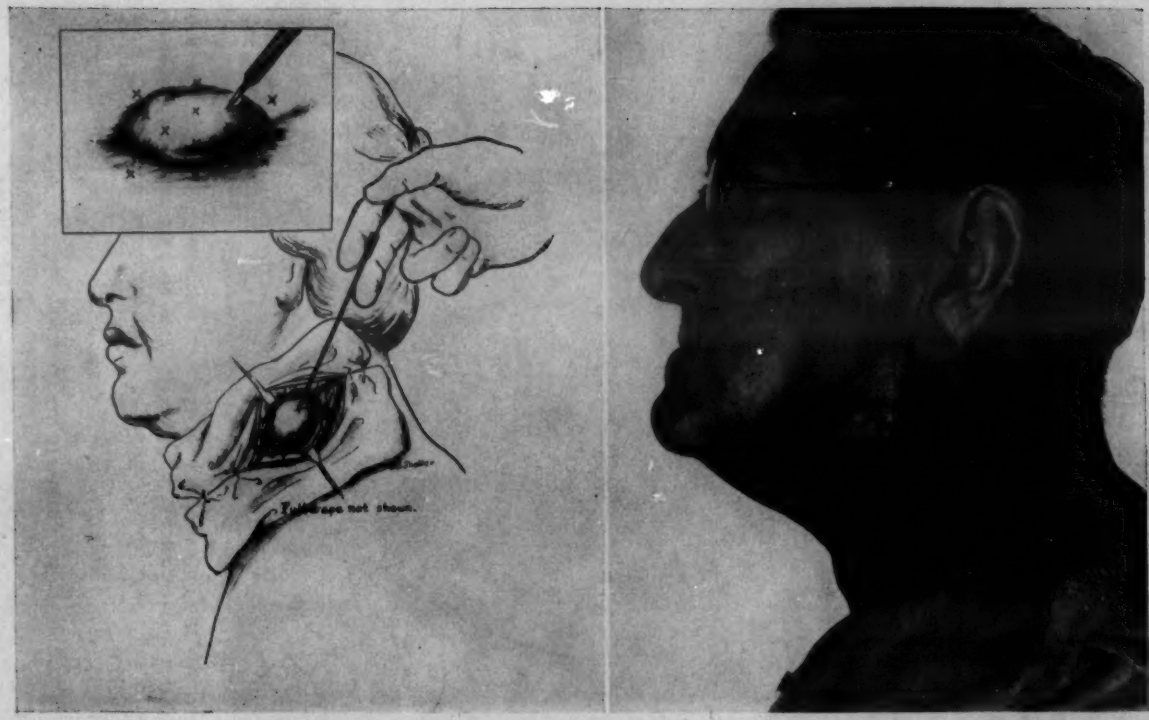


Fig. 5.—At the left is seen a diagram demonstrating the technic of implantation of radon seeds for interstitial irradiation of metastases to the nodes of the neck. At the right is seen a healed incision following dissection of the neck and implantation of radon seeds immediately after completion of a series of high voltage roentgen ray treatments by protracted fractional technic.

prognosis will be most favorable in cases of the better differentiated, slower growing, lower grade tumors, regardless of the method of treatment. Furthermore, we believe that "grading" should be taken into consideration, especially in the selection of treatment for borderline lesions. In cases of the less differentiated, "higher grade" tumors one should feel inclined toward more radical surgery or irradiation. The latter tumors, nevertheless, are to be regarded as carrying a poor prognosis with any method of treatment. In a recent review of cases of patients treated by irradiation alone,[3c] 95 per cent of patients with grade I tumors, 70 per cent with grade II tumors and only 40 per cent with grade III tumors survived five years or more. There were not many patients with grade IV tumors, but they showed a slightly better result than those with grade III tumors.

SUMMARY

The choice of laryngofissure, laryngectomy or irradiation in the treatment of cancer of the larynx must be based primarily on the prospect of cure, and secondarily on conservation of function. Our criteria for the selection of treatment in a given case are: (1) the presence or absence of cervical metastases; (2) the location and extent of the lesion; (3) the motility and mobility of the cord; (4) the histologic character of the lesion, and (5) the general condition of the patient.

Some of the decisions in the selection of treatment are easy to make. For example, the indications for laryngofissure are now fairly clearcut and well established. On the other hand, it is admittedly difficult to establish an arbitrary basis for choosing between laryngectomy and irradiation. In this brief paper an attempt is made to state the pertinent facts gleaned from our experience in regard to these problems.

255 South Seventeenth Street.

LESIONS OF THE CONDUCTION APPARATUS

H. B. PERLMAN, M.D.
CHICAGO

A great variety of threshold curves are found on audiometric examination of patients with lesions of the conduction apparatus. The old teaching that loss of hearing acuity for low frequencies means a conduction lesion and loss for high frequencies a nerve lesion is found to be incorrect from experience with the audiometer, tuning fork tests and loudness balance studies. Present refinements in testing auditory thresholds through most of the acoustic spectrum for both air-conducted and bone-conducted sounds have increased more rapidly than knowledge of the function of the normal and the diseased sound-conducting mechanism itself in response to the various frequencies of the acoustic spectrum. One is confronted with the fact that one cannot predict the audiogram by objective examination of the ear because it is not known what the various components of the conducting apparatus contribute to the hearing curve and how this is altered by disease. Conversely, one cannot look at an abnormal audiogram and accurately define the lesion in the conduction apparatus producing it. This lack of knowledge interferes with the development of rational treatment directed toward conservation or improvement of function. There are a number of reasons why it has been difficult to get accurate information about this problem. First, the clinician who makes the threshold determinations can visualize only a small portion of the structures in the middle ear that are concerned with sound conduction. Polvogt[1] circumvented this difficulty by studying the histopathologic changes of the entire conducting apparatus of subjects who had a normal audiogram shortly before they died. This study showed that a variety of pathologic changes were not detrimental to function. Second, in most pathologic ears a multiplicity of factors operate to influence function. Third, animal experiments in which discrete lesions can be studied are handicapped by the lack of a perfect method of testing auditory function. Fourth, the great body of recently developed knowledge of acoustic physics has not been adequately applied to the study of the conducting mechanism, although the mechanism itself must obey the physical laws of acoustics. Last, the amplitude of movement of the structures is so small that it is difficult to comprehend, let alone measure. For example, at 3000 cycles sound is sensed when the malleus is moved through a distance less than the diameter of a hydrogen molecule. In the laboratory of the division of otolaryngology the movement of the conducting mechanism is being visualized with stroboscopic light and studied both in the living patient and in fresh temporal bones by means of a loud 128 cycle tone.

Careful observation in the clinic and laboratory with a good audiometer in a quiet room recording on regular audiometer charts gives information which, though not ideally accurate, is nevertheless readily applicable to the problems encountered by the clinical otologist. In this way one can learn what the conduction apparatus contributes to the normal threshold curve by observing how this curve is altered when specific lesions are made.

From the Division of Otolaryngology of the University of Chicago.

This paper was read in part at a meeting of the Illinois State Medical Society on May 19, 1942.

1. Polvogt, L. M.: Histologic Variations in the Middle and Inner Ears of Patients with Normal Hearing, Arch. Otolaryng. **23**:48-56 (June) 1936.

One may begin by looking at the original sensitivity curve of the human ear in order to point out what is not apparent in the regular audiometer chart, namely, that the ear is most sensitive to frequencies between 1000 and 3000 cycles (fig. 1). What part do the various components of the external and the middle ear play in producing this area of increased sensitivity? One of the physical factors pertains to the resonance frequency of the external auditory canal. This is about 3000 cycles and theoretically should, like any resonant chamber, increase the intensity of sounds at this frequency which reach the ear drum and thus contribute to the particular acuity of hearing for sounds of about 3000 cycles. However, clinical observation does not bear this out. Patients with almost complete occlusion of the canal by serumen or osteoma or with a large canal as a result of disease or surgical intervention have no appreciable change in the threshold curve for even this range of frequencies, in spite of the changes in the volume of the

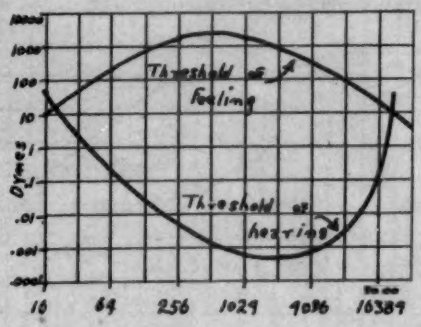


Fig. 1.—Threshold curve, showing region of maximum sensitivity between 1000 and 3000 cycles. This threshold curve is reversed and flattened into a straight line in the clinical audiogram.

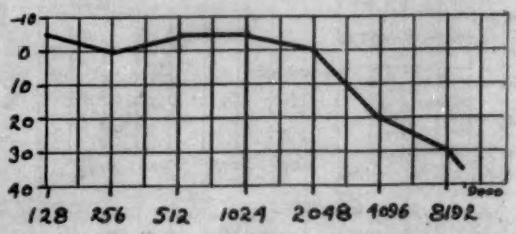


Fig. 2.—Loss for high tones due to obstruction of the external auditory canal by a petrolatum cotton plug in the canal.

external canal with the resultant change in the resonance frequency. Only when complete occlusion of the canal occurs. with obvious interference with the movement of sound waves to the drum, is the hearing impaired. A very small defect in this occlusion, however, permits normal propagation of sound to the drum. Complete occlusion produces a form of conduction deafness, but it is interesting to find that cerumen or a petrolatum cotton plug will interfere more with the propagation of high frequencies than with that of low frequencies (fig. 2).

At 10000 cycles per second the wavelength of sound in air is about 3 cm. At 25 cycles it is over 115 cm. The small diameter of the drum membrane as compared with the wavelengths of audible sound even at high frequencies permits the drum membrane to respond as a unit to all audible frequencies and not to dissipate and distort the stimulus by vibrating in segments or nodes. The cone-shaped contour of the drum membrane increases its rigidity over that of a flat diaphragm

of equal thickness. This increased rigidity also favors its efficient action as a single driving unit for the ossicular chain.

Other factors remaining constant, the response of any structure to sound waves is controlled largely by three entirely different properties: mass, tension or stiffness, and resistance or damping. If the mass of a vibrating structure is increased, the frequency of its natural vibration will be reduced. If the stiffness or tension of the structure is increased, the frequency of its natural vibration will be increased. If the resistance to its motion is increased, the vibration of the structure will be reduced at the resonance frequency and will die out more rapidly; that is, the damping will be greater. For most sounds the conducting mechanism, particularly the drum, malleus and incus, acts as a single unit rotating around an axis passing through the neck of the malleus from the fan-shaped ligament of the incus to the anterior ligament of the malleus. One may profitably consider the stiffness, mass and resistance of the conducting mechanism as a whole with respect

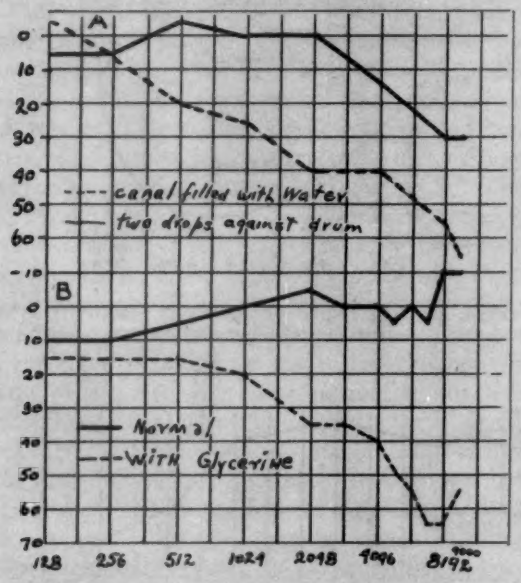


Fig. 3.—Conduction loss for high tones due to increase in mass and damping of the conduction apparatus produced (*A*) by water against the drum and (*B*) by 2 drops of glycerin against half the drum.

to the sensitivity characteristics of the ear. The drum and ossicular chain have a very small mass. The drum membrane is exceedingly thin, often translucent, while the ossicles have a total weight of only 0.05 Gm., and most of this mass is nullified by being delicately balanced around the axis of rotation. This favors the response of the conducting mechanism as a whole to high, as well as to low, frequencies. If the mass and resistance of the conducting mechanism are increased without alteration of its tension, its response to high frequencies should be affected first if the mechansim obeys the laws of acoustic physics. One can increase the mass and resistance, or damping, of the conducting mechanism by loading the anteroinferior half of the drum membrane with a few drops of water or glycerin. This should interfere with the ability of the conducting mechanism to react to sounds of high frequency and the threshold curve verifies this (fig. 3). This holds true whether the fluid is against the external or the internal surface of the drum membrane and explains the loss for high frequencies often seen in

cases of exudative catarrh (fig. 4). The amount of loss for high tones produced by weighting the drum with water, glycerin and mercury varies as a result of differences in their ability to alter the mass and damping. This depends

Fig. 4.—Conduction loss for high tones due to increase in damping and mass of the conducting mechanism produced by fluid in the middle ear (exudative catarrh).

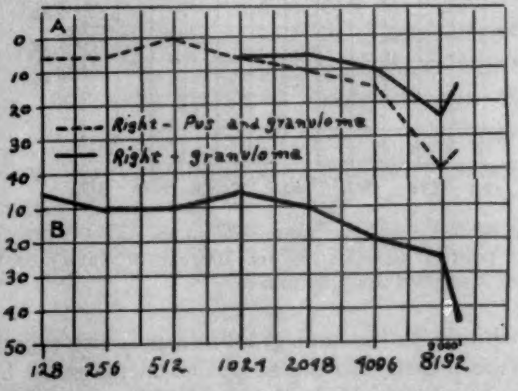


Fig. 5.—Conduction loss for high tones due to increase in mass and damping of the conducting mechanism produced (*A*) by pus against the drum and a granuloma on the drum, and (*B*) by a hemorrhagic bulla on the drum.

on specific gravity, and particularly on the varying ability of the fluid to adhere to the drum membrane. Pus against the drum membrane or a large granuloma

on the membrane as in cases of chronic myringitis, may give a similar audiogram (fig. 5 *A*). Bullous myringitis causes a similar loss for high tones (fig. 5 *B*). If, on the other hand, the tension or stiffness of the conducting mechanism is increased, the mass and damping remaining unchanged, there should, according to laws of acoustic physics, be interference first with the mechanism's response to low frequencies. This situation prevails when there is inequality of air pressure on the two sides of the drum membrane. All have experienced the effect of this inequality on the hearing when driving through the mountains and when flying. One can study

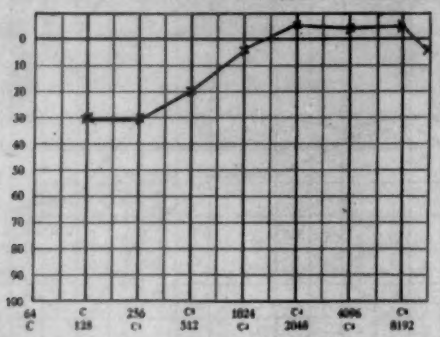


Fig. 6.—Audiogram made with the subject in a pressure chamber, the pressure in the middle ear being 730 mm. of mercury and the external pressure against the drum 760 mm. of mercury. There is loss for low tones due to a pure lesion of stiffness of the drum and ossicular chain.

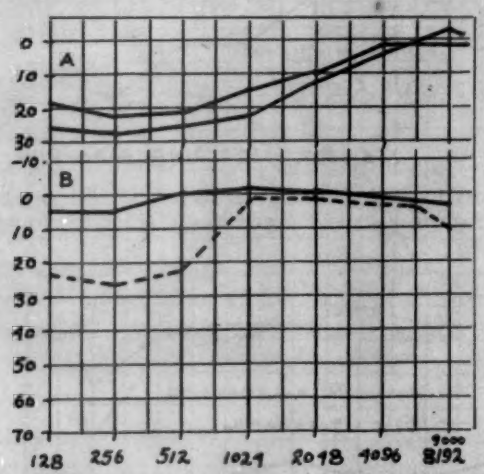


Fig. 7.—Conduction deafness for low tones due to increased stiffness of the conduction mechanism produced by positive pressure (50 and 100 cm. of water) on the drum. *A*, after van Dishoeck [2]; *B*, after Campbell and Hargraves.[4]

the effects of this condition on the audiogram in a pressure chamber (fig. 6) or by artificially changing the pressure in the air sealed in the canal by a tightly fitting audiometer ear piece. To insure normal action of the audiometer, the diaphragm of the receiver must be perforated so that the changed pressure prevails on both sides of the receiver diaphragm. Van Dishoeck [2] used the latter method to study quantitatively the changes in threshold with a measured change in tension, or

2. van Dishoeck, H. A. E.: Negative Pressure and Loss of Hearing in Tubal Catarrh, Acta oto-laryng. **29**:303-312, 1941.

stiffness, of the conducting mechanism due to both positive and negative pressure on the drum membrane. Low tone deafness was consistently produced and varied directly with the amount of change in pressure (fig. 7 *A*). Similar observations were made by Fowler in 1920, using tuning forks.[2a] Maintenance of an equality of pressure on both sides of the ear drum to insure normal stiffness of the conducting mechanism is therefore important for its normal function at low frequencies. Recent experimental observations on animals by Wever and associates [3] have shown this also. This condition of increased stiffness of the conducting mechanism is seen as a clinical entity sometimes after rapid descent in an airplane.[4] Air at reduced pressure of high altitudes is trapped in the middle ear by a blocked tube, while the external surface of the drum membrane is exposed

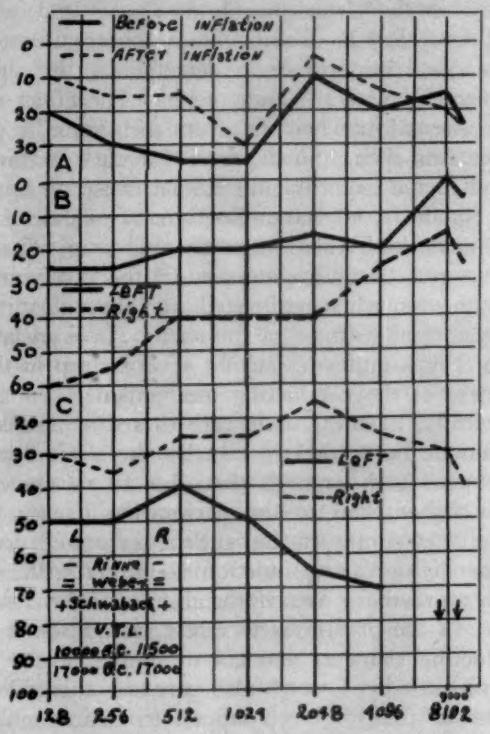


Fig. 8.—Conduction deafness for low tones due to increased stiffness of the conducting mechanism as a result of (*A*) chronic tubal obstruction and (*B*) beginning ossification of the annular ligament (?).

C, radical mastoidectomy cavities, showing (left) skeletonized oval and round windows, with good acuity for air-conducted sound, and (right) obliteration of oval and round windows by connective tissue, with poor acuity for air-conducted sound.

to the air pressure prevailing at ground level. This results in a uniform pressure against the surface of the drum membrane, increasing the tension, or stiffness, of the drum and ossicular chain but not altering their mass. This is reflected in

2a. Fowler, E. P.: Drum Tension and Middle Ear Air Pressures: Their Determination, Significance and Effect upon Hearing, Ann. Otol., Rhin. & Laryng. **29**:688, 1920.

3. Wever, E. G.; Bray, C. W., and Lawrence, M.: The Effect of Middle Ear Pressure upon Distortion [fig. 6], J. Acoustic. Soc. America **13**:182-187 (Oct.) 1941.

4. Campbell, P. A., and Hargreaves, J.: Aviation Deafness, Acute and Chronic, Arch. Otolaryng. **32**:417-428 (Sept.) 1940.

the threshold curve as a low tone loss (fig. 7*B*). This can be reproduced in the pressure chamber (fig. 6). Less acute tubal obstruction will result in a similar audiogram (fig. 8*A*), even when the subject does not leave the ground, and as a result of reduction in pressure of the air in the middle ear. These specific effects of altering the stiffness, mass and resistance of the conducting mechanism explain the threshold curves obtained by Lüscher [5] in his recent experiments.

A factor considered by the acoustics physicist in the construction of a microphone or receiver is the cushion of air behind the vibrating diaphragm. This air cushion has a certain stiffness value when reacting to the rapid changes in pressure of sound waves and is a factor controlling the amplitude of displacement of the vibrating element. The air cushion of the middle ear behind the drum membrane has a volume of about 2 cc. and experimentally is shown to act as a stiffness factor, resisting displacement of the drum membrane by sound waves at very low frequencies (under 100 cycles).[5a] From clinical observations, however, we find that this is not of practical importance in determining the shape of the normal sensitivity curve as recorded with the audiometer. The effect of this confined air cushion in the middle ear is lost when the drum membrane is perforated or when the eustachian tube remains open. Small, clean traumatic perforations of the drum membrane in man and in the experimental animal cause no appreciable change in the threshold curve. Similarly, normal audiograms are obtained from patients with a continuously open eustachian tube when the masking effect of respiration is eliminated. The connective tissue architecture of the drum membrane enables it to retain normal tension even when perforated, and the uninjured ligaments of the ossicular chain insure normal stiffness of the chain. Loss of large amounts of the drum membrane results in a uniform, but not severe, drop in the threshold curve, since mass and stiffness of the conducting mechanism taken as a whole are not greatly altered. Clinically, however, it is rare to see a pure lesion of the drum, such as a clean traumatic perforation. Usually the perforation is the result of suppuration, and the poor audiogram is then due to an associated lesion of the conduction apparatus rather than to the perforation itself. Clinical experience has shown that the value of simply closing such a perforated drum membrane by a prosthesis as a means of increasing function is limited. When improvement of function is obtained by a prosthesis even in the absence of the conducting mechanism, it is due to the ability of the prosthesis to affect restoration in tension, mass and damping of the conducting elements and not to simple closure of the perforation. Recent animal experiments by Lowy [6] also stressed this point. Clinically one encounters retractions or perforations confined to Schrapnell's membrane (pars flaccida) even in cases of suppuration in the mastoid with a normal threshold curve. Usually such lesions are external to the conducting mechanism; that is, they are isolated from the cavity and ossicular chain of the middle ear. A very small degree of thickening of the drum membrane by plaques or small granulations, such as follows infection or thinning of part of the drum membrane, as in cases of healed perforations, is not detrimental to function. Adhesion of a small part of the drum membrane to the promontory, even to the niche of the round window, is compatible with a normal audiogram. With an intact ossicular chain, occlusion of the niche of the round window to a large extent by connective tissue is also compatible

5. Lüscher, E.: Effect of Experimental Disturbances of Tympanic Membrane in Hearing Capacity, Acta oto-laryng. **27**:250-267, 1940.

5a. Békésy, G.: Zur Physik des Mittelohres und über das Hören bei fehlerhaften Trommelfell, Akust. Ztschr. **1**:13-23, 1936.

6. Lowy, K.: Experiments in the Pellett-Type of Artificial Drum, J. Acoust. Soc. America **13**:83-88 (April) 1942.

with a normal audiogram. On the other hand, Polvogt[1] observed that the epitympanic space, where the incudomalleolar articulation is located, was always free of connective tissue bands whenever normal hearing was present. The anterior ligament of the malleus and the fan-shaped ligament of the incus must be intact to maintain normal function at the axis of rotation. Examination of the epitympanic area of a fresh temporal bone with stroboscopic light while the mechanism is driven by a low frequency sound of considerable intensity enables one to see why the epitympanum must be free of any lesion for the ear to retain normal acuity to air-conducted sounds.

Experimentally and clinically any interference with the continuity of the ossicular chain promptly and severely reduces the response of the ear to air-conducted sound and depresses the threshold for the entire acoustic spectrum. One may sever the joint capsule between the incus and the stapes without reducing function—as long as the incus remains in contact with the head of the stapes, but as soon as this contact is lost acuity for all air-borne sound is greatly impaired. Paralysis of the stapedius muscle, with its loss of tonus observed clinically in some cases of paralysis of the facial nerve, has no effect on the threshold curve.[6a] Similarly, patients with paralysis of the motor division of the fifth nerve have normal threshold curves. A small focus of ossification in the annular ligament may be looked on as causing a slight increase in the stiffness of the conducting system and probably manifests itself first in the audiogram as a loss for low tones (fig. 8*B*). Increased stiffness of the ossicular chain due to firm adhesions is probably reflected in the audiogram in a similar way. But freezing of the foot plate of the stapes by any further ossification of the annular ligament (as in cases of well advanced otosclerosis) increases the stiffness of the system so much that it markedly and uniformly reduces the hearing for air-conducted sound. Often in these cases it is possible to demonstrate with a Siegle speculum that the malleus does not move while the drum membrane is massaged. Mobility of the round window membrane is then probably responsible for the acuity for air-conducted sound that remains, and more certainly for preserving the acuity for bone-conducted stimuli. Experimentally it can be shown that if both labyrinth windows are frozen (immobilized with hard plaster of paris), hearing by air conduction as well as by bone conduction appears to be completely destroyed, even though the end organ is normal. Bony closure of the windows has been reported in cases of endemic cretinism and of severe otosclerosis.

In the absence of the conducting mechanism, if the windows are obstructed by only a soft substance, such as connective tissue, fluid or mercury, acuity for bone-conducted sound is retained, although that for air-conducted sound may be severely depressed. If the drum, malleus and incus are destroyed either by suppuration or by surgical intervention, skeletonization of the labyrinth windows is important in retention of acuity for air-borne sound. This is especially true of the oval window niche, which readily becomes obliterated with infection or after operation. If the niche remains clean, with the crura and foot plate of the stapes free of connective tissue, useful acuity for air-conducted sound may be retained even though the rest of the conducting mechanism is gone (fig. 8*C*).

When good acuity for air-conducted sound is present in a chronically suppurative ear it is reasonable to assume that the ossicular chain is not destroyed or irreversibly diseased, and this fact should be severely respected in consideration of treatment. When local treatment fails to dry such an ear, a simple mastoidectomy may sometimes bring about the desired result, even after years of discharge, and assure

6a. Perlman, H. B.: Hyperacusis, Ann. Otol., Rhin. & Laryng. **47**:947-953 (Dec.) 1938.

retention or improvement of function. It may be wiser in some cases to leave an ear wet with good function than to dry it up by destroying the conducting mechanism in a radical mastoid operation, with the additional likelihood of further impairment of function due to obliteration of the oval window niche by connective tissue. Patients with perforation of Schrapnell's membrane and cholesteatoma of the attic and antrum retain their good auditory function when the posterior wall of the canal is taken down without interference with the ossicular chain or the drum membrane.

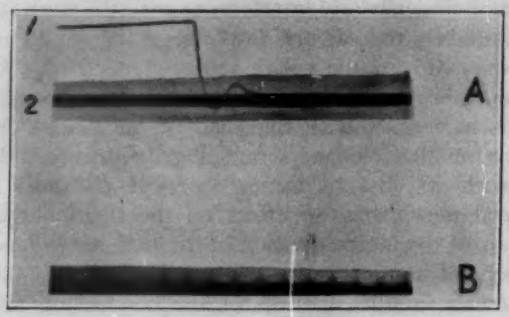


Fig. 9.—Record of the natural oscillation of the human conduction apparatus (taken from a mirror on the head of the malleus). (*A*) The apparatus was displaced within its elastic limits toward the negative side of center (*1*) and allowed to fall back to its resting state (*2*) in its own manner. The natural frequency of the conduction apparatus is seen to be about 700 cycles per second, and the large degree of damping is apparent. (That is the rapidity with which the mechanism comes to rest.) *B* is a 1000 cycle timing wave.

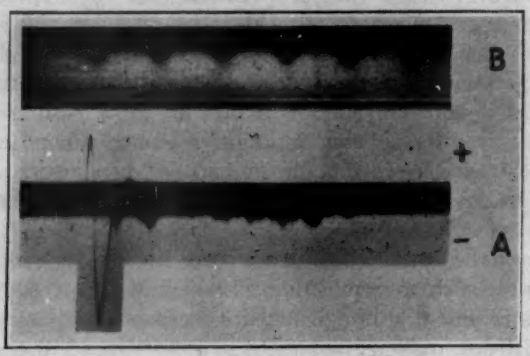


Fig. 10.—*A*, response of the human conduction apparatus to a single stimulus of a shock pulse. Only one major vibration is induced. This illustrates the high degree of damping or the abruptness with which the mechanism comes to rest after a single displacement. The wave above the line is positive; the one below the line is negative. Note the greater amplitude of the negative wave. *B* is a 1000 cycle timing wave.

The conduction mechanism, coupled to the labyrinth and immersed in the air chambers of the middle and external ears, has a resonance frequency (fig. 9). In man this is about 700 cycles per second, and in some way this is reflected in the sensitivity curve, although this curve shows no sharp peak of sensitivity corresponding to such a resonance frequency. One reason for this is that the conducting mechanism when set into vibration by a single pulse rapidly comes to rest; that is, it is highly damped (fig. 10). This flattens and widens the resonance curve greatly and

prevents excessive and prolonged stimulation of the end organ at the resonance frequency and at all frequencies (fig. 11). Furthermore, the vibration of the end organ immersed in fluid is even more highly damped. The mechanism is therefore able to respond accurately to a continuous stream of acoustic disturbances and to stop reacting immediately after cessation of the stimulus. An increase in the mass of the conducting mechanism will reduce its resonance frequency. Impairment of hearing for high frequencies under this condition can be explained by taking into account the normal sensitivity curve of the ear and the damping. When this change in the mechanism is accompanied by increase in damping, the loss for high frequencies is even more pronounced. An increase in the degree of resistance or damping of the mechanism will of itself cause impairment in the response of the mechanism to the high frequencies. Conversely, an increase in stiffness of the conducting mechanism will raise its resonance frequency and impair the response of the mechanism to low frequencies.

In clinical experience with the deaf one finds that, depending on occupation, intelligence, etc., people vary greatly in the demands they make on their auditory sense and the amount of use they can make of the sound that they hear and of lip reading, so that one cannot predict the degree of handicap and suffering of the

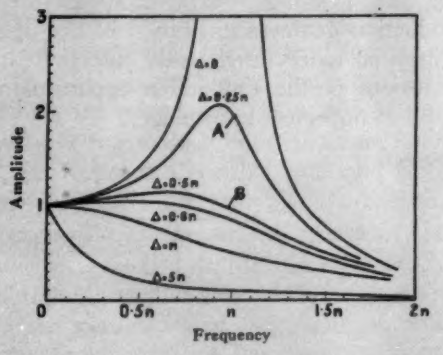


Fig. 11.—Forced vibration. Relation between frequency and amplitude of displacement for various degrees of damping (from Davis, A. H.: Modern Acoustics, New York, The Macmillan Company, 1934). To prevent overamplification at the resonance frequencies, *n* (*n* equals 700 double vibrations for the conduction mechanism of the ear), a high degree of damping is essential. The conducting system of the ear approaches this, in a curve between *A* and *B*.

patient by looking at a threshold curve alone. Moreover, one must never lose sight of the fact that the most important single use of hearing in man is for understanding of the spoken voice. The different frequencies tested for in the audiogram vary greatly in their importance with respect to the hearing of the spoken voice, and this is taken into account in construction of deafness disability tables (Sabine [7]; Fowler [8]). In general hearing acuity is greatest through the frequencies found in the acoustic spectrum of speech. But it is particularly important that every one hear well at frequencies of 1000 or 2000 cycles. The old-fashioned telephone transmitter carried only a small portion of the acoustic spectrum of speech (frequencies of about 1000 cycles). This contraction and distortion of the speech spectrum by the telephone did not greatly affect intelligibility of the spoken voice, and many controlled experiments in the Bell Telephone Laboratories have shown this. It is

7. Sabine, P. E.: On Estimating the Percentage Loss of Useful Hearing, Tr. Am. Acad. Ophth. (1941) **46**:179-187 (March-April) 1942.

8. Fowler, E. P.: A Method for Measuring the Percentage of Capacity for Hearing Speech, J. Acoust. Soc. America **13**:373-382 (April) 1942.

important to remember that the increased acuity of one's auditory sense to stimuli between 1000 and 3000 cycles per second is expressed not only by the smaller amount of physical energy of the acoustic stimulus needed to induce a threshold response but in other significant ways. Through this part of the acoustic spectrum one can sense differences in loudness with smaller changes in the physical intensity of the stimulus. Through this frequency range one can detect differences in pitch with smaller changes in the frequency of the stimulus. Furthermore, a greater number of loudness steps are sensed through this range of frequencies because the ear tolerates more steps of intensity before the sound becomes painfully loud. When one reviews the observations on the physiology and pathology of the conducting mechanism, one finds little to explain these characteristics of special sensitivity of the auditory sense in this area of the acoustic spectrum. Observations by Fowler[8] on pitch discrimination in deafened persons partly confirm this. The explanation probably lies somewhere in the neural mechanism. Ability to understand speech and to comprehend its meaning while only a small part of its acoustic spectrum in this range of frequencies is reaching the ear and to separate this portion from the acoustic spectrum of background noise, or even of other speakers, is probably due to the action of the higher cortical centers subserving audition in the process of listening.

Audiograms for conduction deafness examined in the light of the factors considered in this report may be more intelligently interpreted, but many threshold curves associated with lesions of the conduction apparatus are, nevertheless, still difficult to explain because of imperfect knowledge.

950 East Fifty-Ninth Street.

RELATION OF HEMATOLOGY AND OTOLARYNGOLOGY

GOODLATTE B. GILMORE, M.D.
NEW YORK

Examination of the blood of persons with some otolaryngologic diseases yields important information relative to diagnosis or prognosis. There are pathologic conditions in the blood which reveal themselves by lesions in the mucous membrane of the nose and throat. Conversely, diseased conditions in the nose and throat produce changes in the blood with which the otolaryngologist should be familiar.

The blood is a highly complex fluid which holds corpuscles in suspension. When blood is centrifuged the cells are separated from the fluid portion, called the plasma. The latter contains proteins, various organic and inorganic materials, hormones, antibodies, enzymes and chemical compounds whose elements are still undetermined.

The cells of the whole blood are the red cells, or erythrocytes, the white cells, or leukocytes, and the platelets, or thrombocytes.

According to Best and Taylor,[1] the chief function of the erythrocytes is to store and carry hemoglobin. Hemoglobin in turn is the principal collector and distributor of oxygen throughout the body. If the hemoglobin is partially destroyed the plasma, a poor substitute, attempts the added duty as a vehicle for carrying oxygen.

The leukocytes consist of two main groups, the nongranulocytes and the granulocytes. The nongranular leukocytes are the small and large lymphocytes and the monocytes. The granulocytes are the eosinophils, the basophils and the neutrophils.

The white blood corpuscles are important agents in the defense of the body against invading organisms. This protection depends on the phagocytic action of the neutrophilic polymorphonuclear leukocytes.

The blood platelets are non-nucleated fragments of protoplasm originating in the cytoplasm of the megakaryocytes. When platelets disintegrate, thromboplastin is liberated, and this substance plays an important role in the complex biochemical reaction that takes place in the clotting of blood.

In postnatal life the erythrocytes, granulocytes and platelets are manufactured in the red bone marrow. Lymphocytes originate in lymphoid tissue. While new blood elements are constantly fabricated, the older cells are destroyed. The spleen is particularly active in this destruction. The normal balance of a healthy person depends on the interchange of the new blood elements for the old effete cells.

Some diseases having otolaryngologic manifestations show an increase in the cellular elements of the blood. This increase occurs in leukemia, infectious mononucleosis, sepsis, polycythemia and Vincent's angina. In purpura, granulocytopenia and Plummer-Vinson syndrome a decrease exists in the cellular elements. Hemophilia and congenital multiple telangiectasia fall into a third group, distinguished by a normal number of blood cellular elements, except as they are influenced by hemorrhage. In hemophilia the coagulation time is abnormal, and in congenital multiple telangiectasia the vessels in the characteristic lesions have very thin walls.

Read before the Bronx Otolaryngological Society, Nov. 25, 1941.
From the Otolaryngological Department, Morrisania City Hospital.
Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., June 1942.

1. Best, H. C., and Taylor, N. B.: The Physiological Basis of Medical Practice, ed. 2, Baltimore, Williams & Wilkins Company, 1940, pp. 1-181.

Ordway and Gorham [2] stated: "The four outstanding landmarks causing one to suspect that an unknown condition in any particular person may be a disease of the blood are, (1) anemia, (2) enlargement of the lymph glands or spleen, (3) hemorrhage into the tissues or from the mucous surfaces, and (4) throat lesions, particularly of the membranous or ulcerative type, varying greatly in degree, extent and type." A moment's reflection shows one how often otolaryngologists are confronted with one or more of these findings.

LEUKEMIA

Blood dyscrasias are not true diseases of the blood itself. The cause is some known or unknown toxin which stimulates or depresses the hemopoietic system. To this the leukemias are no exception, each variety being characterized by an extraordinary increase in a certain type of white cell. A typical blood cell count in leukemia exhibits hundreds of thousands of white corpuscles per cubic millimeter, the differential diagnosis being dependent on the preponderating type of cells present. The leukemias are: acute lymphatic leukemia, chronic lymphatic leukemia, acute myeloid leukemia, chronic myeloid leukemia and acute monocytic leukemia.

Epistaxis, persistent and repeated, can be the initial sign of leukemia. Cases are recorded in which the blood lost had a peculiar waxy white appearance. Love [3] stated that in leukemia there may be hemorrhage in the external meatus, tympanic membrane, middle ear and labyrinth. Of his series of 153 cases, in 39 vertigo, tinnitus or deafness was complained of. This is attibuted to a leukocytic infiltration or hemorrhage involving the eighth nerve or its end organ.

The bizarre lesions of leukemia are frequently found in the oral cavity. Bleeding gums resembling those of Vincent's infection and lesions imitating scurvy and stomatitis are common. Necrotic or membranous areas may occur in various localities throughout the mouth.

INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis, or "glandular fever," was first described by Pfeiffer in 1889.[4] It appears in sporadic, endemic or epidemic form. Almost without exception young adults are attacked. Heck [5] mentioned five minor varieties, each of which runs a febrile course but which differ in the degree of glandular involvement. Angina is a more or less constant symptom, and cervical adenopathy is always out of proportion to the extent of the lesion in the throat.

Early in the disease the blood picture is normal. Later the leukocyte count rises, but the red blood cell count, the hemoglobin content and the platelet count remain stationary. The white blood cell count showed as high as 87 per cent lymphocytes in 1 case reported by Grove.[6] This was accompanied by a drop in the neutrophils to 13 per cent. This depression in the percentage of neutrophils is constant in infectious mononucleosis and may lead to the mistaken diagnosis of granulocytopenia. Basophilic lymphocytes with indented, lobulated or kidney-

2. Ordway, T., and Gorham, L. W.: The Diagnosis and Treatment of Diseases of the Blood, New York, Oxford University Press, 1938; cited by Love.[3]

3. Love, A. A.: Manifestations of Leukemia Encountered in Otolaryngologic and Stomatologic Practice, Arch. Otolaryng. **23**:173 (Feb.) 1936.

4. Pfeiffer, cited by Heck.[5]

5. Heck, F. J.: Handbook of Hematology, New York, Paul B. Hoeber, Inc., 1938, vol. 4, p. 2583.

6. Grove, W. E.: Laryngologic Aspects of Sporadic Infectious Mononucleosis, Arch. Otolaryng. **32**:472 (Sept.) 1940.

shaped nuclei and a foamy-appearing cytoplasm, due to vacuolation, are commonly seen. This blood picture while characteristic is not pathognomonic.

Paul and Bunnell [7] in 1932 demonstrated a heterophilic antigen in the blood of patients suffering from infectious mononucleosis. This is identical with the reaction observed in the blood of persons with horse serum sickness, an acquired ability of the blood to agglutinate sheep red blood corpuscles when it is highly diluted. Absence of a history of serum sickness is therefore necessary before one may accept the finding in the diagnosis of infectious mononucleosis.

Davidsohn [8] removed all the agglutinins for sheep red cells by treating the patient's serum with beef red cells and 50 to 75 per cent by treating it with guinea pig kidney emulsion. This supplementary test confirms the diagnosis.

Grove [6] advised caution if sulfanilamide is used in treatment of this disease. The neutrophil percentage is low in patients with infectious mononucleosis; so the administration of this drug, given with the assumption that the angina is due to streptococci, from its occasional toxic action on the neutrophils, may confuse the issue. He also stated that the granulocyte count can be forced down to a dangerous level by sulfanilamide.

SEPSIS

In sepsis the white blood cell and differential counts should be studied by the modern Schilling hemogram. Arneth was the first to call attention to the fact that the number of lobes in the nucleus of any neutrophil depends on the cell's age, the older cells having the larger number. According to the Schilling index, four such age groups are recognized: young myelocytes with a single spherical nucleus; young metamyelocytes with a slight indentation; older metamyelocytes with a distinct lobulation, Schilling band cells or "staff" cells and older neutrophils. Any increase in the percentage of cells of the earlier and younger types is spoken of as a "shift to the left." Sepsis and pyogenic infections stimulate the bone marrow to a greater production of white cells and cause this "shift to the left." In pernicious anemia there is an increase in the percentage of older white cells. This is known as a "shift to the right."

The Schilling hemogram, showing the extent of the "shift to the left," estimates the severity of the infection in sepsis. When repeated at intervals it gives important prognostic data. A definite return to normal or relative "shift to the right" will be noted before other signs of improvement are observed.

Secondary anemia is constant in hemolytic streptococcic bacteremia. This anemia is caused by the action of the hemolytic substances excreted by the organisms, which attack the erythrocytes and liberate free hemoglobin.[9] To combat the anemia and declining red cell count, transfusions of compatible whole blood are given. These transfusions restore the necessary cellular elements, renew the serum and plasma volume and rid the system of much toxic material. If compatible blood from a donor recently recovered from a similar infection is obtainable, better results can be anticipated. Large amounts are unnecessary. Small transfusions (150 to 250 cc.) repeated every few days yield better results. The object of this form of therapy is to hold the hemoglobin well above 60 per cent.

Jansky, Moss and Landsteiner proved that the blood of any person fits with precision into one of four groups, depending on its agglutinating reaction.

---

7. Paul, J. R., and Bunnell, W. W.: The Presence of Heterophile Antibodies in Infectious Mononucleosis, Am. J. M. Sc. **183**:90 (Jan.) 1932.

8. Davidsohn, I.: Serological Diagnosis of Infectious Mononucleosis, J. A. M. A. **108**: 289 (Jan. 23) 1937.

9. Sutherland, J. M.: Transfusions of Blood in the Care of Patients After Operations for Otitic Sepsis, Arch. Otolaryng. **30**:876 (Dec.) 1939.

Landsteiner [10] discovered two definite substances in blood serum, which he named isoagglutinins, and two of a similar nature in the corpuscles, isoagglutinogens. The isoagglutinins he indicated by the small Greek letters α and β, and the isoagglutinogens, by the capital letters A and B. When the isoagglutinins are absent a small zero (o) is employed, and a large zero(0) denotes the absence of isoagglutinogens. When two bloods come in contact so that α is present with A (αA) or β with B (βB) agglutination occurs; in other words they are incompatible. Since this could not obtain in vivo there result four groups, 0$\alpha\beta$, Aβ, Bα and AB0, simplified by using only the letters indicating the characteristics of the corpuscles, 0, A, B and AB, as shown in the accompanying table.

The original classification of Jansky and the substitute classification of Moss result in confusion. Grouping the blood according to its serologic character as recommended by Landsteiner has received universal approval.

As a result of further investigation, Landsteiner and Wiener [11] discovered an agglutinin in rabbit blood which followed injections of blood of the Macacus rhesus monkey. When this agglutinin was used against human blood, a new substance, named by these authors the Rh factor, was detected in the human erythrocytes. The antibody in the rabbit blood they called the anti-Rh agglutinin.

Levine [12] found that 86 per cent of human bloods contain this factor in the red cells and 14 per cent do not. Only the white race has been investigated. Like the

Blood Groups

Jansky	Moss	International	Cells Contain Isoagglutinogens	Serum Contains Isoagglutinins
I	IV	O	O	$\alpha + \beta$
II	II	A	A	β
III	III	B	B	α
IV	I	AB	AB	o

A and B factors of the major blood groups the Rh factor is inherited from the father.

In the light of this discovery Burman [13] explained the pathogenesis of erythroblastosis foetalis. A fetus with blood containing the Rh factor in the uterus of a woman without this factor in her blood can excite the formation of the anti-Rh agglutinin in the mother's blood. This agglutinin will then pass through the placenta and destroy the Rh-containing fetal blood.

It is further believed that anti-Rh agglutinins are produced in a certain percentage of persons who do not have the Rh factor in their blood by repeated transfusions of blood that does contain this substance. This process, whereby the anti-Rh agglutinins are formed either in the blood of the pregnant woman (which does not contain the Rh factor) or by repeated transfusions of blood containing the Rh factor to a patient with blood that does not contain this substance is spoken of as isoimmunization, since it occurs between bloods of the same group otherwise compatable.

10. Landsteiner, cited by Best and Taylor.[1]

11. Landsteiner, K., and Wiener, A. S.: An Agglutinable Factor in Human Blood Recognized by Immune Sera for Rhesus Blood, Proc. Soc. Exper. Biol. & Med. **43**:223 (Jan.) 1940.

12. Levine, P.: The Role of Iso-Immunization in Transfusion Accidents in Pregnancy and in Erythroblastosis Fetalis, Am. J. Obst. & Gynec. **42**:165 (July) 1941.

13. Burman, L.: The Common Etiology of Erythroblastosis and Transfusion Accidents in Pregnancy, Am. J. Obst. & Gynec. **42**:389 (Sept.) 1941.

According to Levine,[12] these anti-Rh agglutinins account for almost 90 per cent of the intragroup transfusion accidents. Because of their presence the donor's blood is hemolyzed and agglutinated, which causes oliguria or anuria with a possible fatal outcome. These accidents are preventable if the new cross matching technic is used, i. e., refrigerating the patient's serum and the donor's cell suspension for thirty minutes prior to low speed (500 revolutions per minute) centrifugation.

The new classification of the major blood groups and a possible intragroup incompatibility are of importance in the otolaryngologic field, where transfusions are so frequently used.

When sepsis is complicated by thrombophlebitis of a venous sinus Schall[14] advocates the intravenous use of heparin plus chemotherapy. He reports cures in 3 cases of septic thrombophlebitis of the cavernous sinus by this method. Howell[15] said that heparin reacts with some constituent in the blood and forms an antithrombin. Its anticoagulant effect maintains the normal fluidity of the blood and prevents the extension of the thrombus. It has no effect on the existing clot. The blood is sterilized by the chemotherapeutic drug.

POLYCYTHEMIA

An erythrocyte count of 7,000,000 or more per cubic millimeter of blood is symptomatic in polycythemia. The oxygen content of the blood is decreased in a few pathologic conditions, and the red bone marrow responds by producing an abnormal number of red cells. This occurs in patients with chronic tracheal stenosis, in victims of chronic carbon monoxide poisoning and in persons suffering from repeated small hemorrhages.

In polycythaemia vera the red blood cell count may reach 14,000,000 to 20,000,000 per cubic millimeter. The total blood volume and its viscosity are raised, and the other components stay within comparatively normal limits. The cause of this disease is unknown. Cyanosis, dyspnea on slight exertion, enlargement of the spleen, epistaxis and bleeding from the gums are characteristic signs. Fitz-Hugh[16] pointed out that patients with this condition are poor surgical risks because of the danger of postoperative thrombosis or of hemorrhage. He also stated that Ménière's syndrome can occur in a patient with this disease because of thrombosis of a vessel in the inner ear.

VINCENT'S ANGINA

This is generally a benign and easily diagnosed malady. In cases in which it is obstinate there may be a hemic response. The mature small lymphocytes predominate over the other forms, reaching as high as 90 per cent in a total count of 30,000 to 40,000. When such an extreme is reached, according to Shea,[17] the prognosis is bad.

PURPURA

Under the title purpura are included a variety of hemorrhagic states with which spontaneous bleeding takes place beneath the skin, from the mucous membrane or into the joints and internal organs. It may be considered a symptom of a blood

14. Schall, L. A.: Treatment of Septic Thrombophlebitis of the Cavernous Sinus, J. A. M. A. **117**:581 (Aug. 23) 1941.
15. Howell, W. H.: Blood Coagulation, J. A. M. A. **117**:1059 (Sept. 27) 1941.
16. Fitz-Hugh, T., Jr.: Some Interrelationships of Modern Hematology and Otolaryngology, Arch. Otolaryng. **34**:421 (Aug.) 1941.
17. Shea, J. J.: The Blood in the Various Anginas, Arch. Otolaryng. **12**:366 (Sept.) 1930.

dyscrasia rather than a disease itself. An abnormal permeability of the capillary walls is present in most cases, permitting red blood cells to escape into the surrounding tissue. In patients with scurvy, with which weakness of the capillary walls exists, petechiae are also a constant finding.

In thrombopenic purpura the thrombocytes are lacking. Normally 200,000 to 400,000 per cubic millimeter, they are either greatly reduced or absent. Bedson stated the belief that platelet reduction must coexist with capillary defects to give the true picture of purpura. He expressed the opinion that the platelets are protectors of the capillary walls and defects in the latter are the outcome of the reduced platelet count.

A frequent initial symptom of purpura is epistaxis. Warren [18] stated that every patient with recurrent nose bleed "should have a complete blood count, bleeding time, clotting time, clot retraction test, platelet count, fragility test, prothrombin time and bone marrow study to make a proper classification." This group of tests should be familiar to the otolaryngologist.

GRANULOCYTOPENIA

Records of this disease appeared in the literature with increasing frequency about 1930. A marked diminution or even absence of adult granular leukocytes in the circulating blood distinguishes granulocytopenia from other blood dyscrasias. Frank [19] stated that a diagnosis is clear only "when the total leukocyte count is below 1,000 per cubic millimeter with a very low count or absence of granulocytes." The red blood cell and platelet counts are usually normal, and hemorrhages do not take place. In aleukemic leukemia the erythrocytes and thrombocytes are reduced and small hemorrhages occur.

Some toxic agent exerting a depressant action on the hemopoietic function of the bone marrow produces this condition. The condition has followed the administration of certain coal tar derivatives, notably aminopyrine, the barbiturates and dinitrophenol. The toxic element of these chemicals is the benzene ring. To date no etiologic bacterial toxin has been discovered.

A natural sequence to the leukopenia in this disorder is an almost total loss of phagocytic ability in the blood. Membranous or ulcerative infections in the mouth or pharynx regularly occur. The atypical peritonsillar abscess should be viewed with suspicion—that is, an abscess from which incision does not liberate pus as expected.

Following the widespread use of sulfanilamide or its derivatives, leukopenia has frequently been reported and occasionally fatal cases of granulocytopenia. This toxic effect may be due to an individual idiosyncrasy or to an overdosage or prolonged administration. Before a patient receives a chemotherapeutic agent a complete blood count should be done to establish a base for later comparison. This should be repeated every forty-eight hours during the time the drug is administered. A sudden drop in the leukocyte count below 4,000 per cubic millimeter or a fall in the hemoglobin content of more than 20 per cent in one day is, according to Osgood,[20] cause for immediate discontinuance of administration of the drug.

PLUMMER-VINSON SYNDROME

This syndrome (anemia, glossitis and dysphagia) borders the fields of otolaryngology and bronchoesophagology. The anemia is the idiopathic hypochromic

18. Warren, E. D.: Thrombopenic Purpura and the Role of X-Ray Treatment to the Spleen, Ann. Otol., Rhin. & Laryng. **49**:453 (June) 1940.
19. Frank, I.: Granulocytic Angina, Arch. Otolaryng. **23**:310 (March) 1936.
20. Osgood, E. E.: Chemotherapy, Arch. Otolaryng. **33**:961 (June) 1941.

type. Kernan [21] cited Thiele and Kühl, who claimed that the disease picture is typical since the diminished hemoglobin value is in proportion to the lowered erythrocyte count and the color index is always reduced. This anemia is frequently associated with achlorhydria, and the diameter of the blood cells is below average.

HEMOPHILIA

Hemophilia is seen in persons having a hemorrhagic diathesis. It is rarely observed in otolaryngology yet is often considered as a possibility when bleeding occurs. Its causation is unknown. It appears only in males who show a typical type of inheritance.[1] The patient's mother transmits the disease but is not a victim. Her female children, while capable of passing the malady on to male descendants, are not affected.

Spontaneous hemorrhages occur from the mucous surfaces in any part of the body in hemophilia. The nose is the most frequent locality.

A greatly prolonged coagulation time is constant in this disorder. The bleeding time is normal. The platelet count is standard, but the platelets have an increased stability and slow disintegration rate. Howell [15] said that all of the agents of coagulation are present in proper amounts in hemophilic blood except thromboplastin. This factor is supplied by the physiologic destruction of the platelets. Their abnormal stability establishes a longer coagulation time and resultant tendency to bleed.

The exact chemical structure of tissue thromboplastin is unknown; so it cannot be prepared synthetically. The parenteral administration of this substance in the therapy of hemophilia is therefore precluded. Reliance must be placed on transfusions to supply the missing element.

CONGENITAL MULTIPLE TELANGIECTASIA

The genealogy of a patient with this disorder may reveal recurrent epistaxis in members of a family through as many as four generations.[22] Unlike hemophilia, this disease affects males and females alike and is transmitted equally. Larrabee and Littman, cited by Houser,[22] showed that there exist many red and purple nevi on the skin, in the oral cavity and on the mucous membrane of the nose. These telangiectatic areas have a tendency to bleed.

No pathologic blood picture exists. A secondary anemia in proportion to the amount of blood lost is noted if hemorrhages occur from one or more of these lesions. Hematologists point to an abnormal thinness of the vessel walls in the nevi to explain the bleeding tendency.

COAGULATION TIME, BLEEDING TIME, SEDIMENTATION RATE

By coagulation time is meant the time consumed by blood in clotting after it has been liberated. Normally this is four to seven minutes. The bleeding time is the time it takes blood to clot firmly enough to stop bleeding from a puncture wound in the skin. The normal for this is two and one-half minutes. These tests prior to tonsillectomy were at one time considered as a vital precaution. McKinney [23] made known his doubts as to their value in lowering the incidence

21. Kernan, J. D.: Plummer-Vinson Syndrome, Arch. Otolaryng. **32**:662 (Oct.) 1940.
22. Houser, K. M.: Congenital Multiple Hemorrhagic Telangiectasia with Epistaxis, Arch. Otolaryng. **20**:438 (Sept.) 1934.
23. McKinney, R.: Coagulation Test, Arch. Otolaryng. **6**:158 (Aug.) 1927.

of postoperative bleeding in 1927. Since then their negative worth has been accepted. A few well chosen questions about the family and personal history have far greater value.

Hume and Kahn [24] called attention to the paucity of contributions on the sedimentation rate in otolaryngology. The test depends on the suspension stability or rate of sinking of the red cells when clotting is hindered by an anticoagulant. The rate varies in health and in disease. These authors conclude that it is increased in direct ratio to the amount of tissue destroyed. This infers that a rising rate could signify progressive destruction of bone. In general, it can be said that acute infections elevate the rate and that in the quiescent stage of chronic diseases this does not take place.

Further evidence should be at hand before too much emphasis is placed on this test in the otolaryngologic field.

SUMMARY

An attempt is made to show the relationship between hematology and otolaryngology by the enumeration of specific examples. Emphasis is laid on the fact that a disorder in one field may manifest itself by lesions in the other. The close association of these branches of medical practice is demonstrated.

Blood dyscrasias, such as the leukemias, purpura, polycythemia, granulocytopenia and hemophilia, have at times otolaryngologic signs that may mislead the observer into overlooking the cause by paying too much attention to the effect.

The changes in the blood in sepsis, Vincent's angina, the Plummer-Vinson syndrome and infectious mononucleosis and the pathologic changes of congenital multiple telangiectasia are discussed.

The use of heparin in thrombosis is mentioned.

The new classification of the four major human blood groups is given. The possibility of intragroup transfusion accidents is explained by the existence of the newly discovered Rh factor and its anti-Rh agglutinin.

Reference is made to the value of the coagulation time, the bleeding time and the sedimentation rate in otolaryngology.

Dr. Clarence H. Smith and Dr. William Aronson made valuable suggestions in the preparation of this thesis.

2940 Grand Concourse.

24. Hume, J. R., and Kahn, H.: Blood Sedimentation in Acute Otitis Media and Its Complications, Arch. Otolaryng. **29**:820 (May) 1939.

INCIDENCE OF VASOMOTOR RHINITIS DURING PREGNANCY

MEADE MOHUN, M.D.
SAN MATEO, CALIF.

The vasomotor congestion frequently observed during the last stages of pregnancy first attracted my attention to the relationship existing between nasal disturbances and conditions in the female reproductive system. My case histories are taken from 1,900 obstetric cases at the San Mateo Clinic. In a certain number of cases difficulty in breathing developed in the eighth or ninth month of pregnancy. In the large percentage of such cases the difficult breathing could not be explained entirely on the basis of interference with diaphragmatic movement and other mechanical causes. There appeared to be an actual primary nasal congestion.

This condition of nasal congestion is seen sufficiently frequently during late pregnancy that the obstetrician usually assures the sufferer that her discomfort is a temporary and almost physiologic development of pregnancy which will not require the services of an otolaryngologist. But the condition varies greatly according to the individual patient. In some cases nasal congestion develops earlier and in a more severe form. These are the cases that come to the attention of the laryngologist.

This paper represents a report of 20 cases which I was able to study somewhat in detail. The accompanying graph shows the development in these cases of marked nasal congestion during pregnancy and the recession following delivery. The line showing the incidence of this condition represents a composite for all my cases. The line showing the rise in estrogen represents the normal physiologic production of this substance during pregnancy as shown by Blaisdell.[1] The dotted line is taken from the article by Mortimer, Wright and Collip[2] and represents the normal physiologic development of increased reddening and swelling of the nasal mucosa as found in a series of examinations.

These 20 cases are described later in the part of this paper dealing with maximum production of estrogen and its related nasal conditions. Most of the article is devoted to the construction of as reasonable a background as possible (by review of the experimental literature) for the facts I have encountered clinically. I realize that a complete reconcilement is not possible.

SUMMARY OF LITERATURE ON THE NASOGENITAL RELATIONSHIP

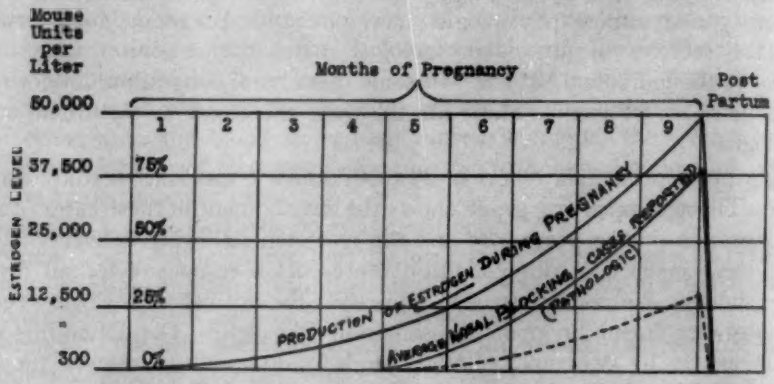
The nasogenital reflex theory dates back to antiquity, as an early recognition of the frequently observed relationship existing between conditions in the nose and events in the genital system, a relationship which has both physiologic and pathologic aspects, and which may be specific, as implied by the old reflex theory, or may be merely the manifestation of a generalized vasomotor or other systemic changes making itself apparent in the vaginal and the nasal mucosa because of the susceptibility of these somewhat similar structures.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1942.

1. Blaisdell, D. H.: Laryngoscope **48**:699 (Oct.) 1938.

2. Mortimer, H.; Wright, R. P., and Collip, J. B.: Canad. M. A. J. **35**:503 (Nov.); 615 (Dec.) 1936; **37**:445 (Nov.) 1937.

In the second of a series of three articles dealing with extensive work in this field, Mortimer, Wright and Collip [2] reviewed some of the older literature, the subject matter of which they divided into three main types: the relation of the nose to dysmenorrhea; the significance of epistaxis during menstruation and pregnancy, and the normal, physiologic nasogenital relation. In addition, in more recent literature, an increasingly larger proportion of attention has been given to the relation between atrophic rhinitis and the genital system. In fact, much of Mortimer, Wright and Collip's work is devoted to this condition and its alleviation through estrogen therapy. The present article adds to this list of relations a consideration of the incidence of vasomotor rhinitis during pregnancy. There is, apparently, a normal, physiologic nasogenital relationship or correlation. Then there is the possibility of events in the genital system having a pathologic response in the nose; likewise, stimulation or irritation of the nasal mucosa might have a pathologic repercussion in the vagina or other part of the genital system. Eggleston, cited by Mortimer,[3] found evidence of an estrogenic effect on the immature mouse uterus of extracts made from the nasal mucosa of the ox. He



Graph showing relation of estrogen production during pregnancy to nasal blocking. The dotted line represents physiologic reddening and swelling of mucous membranes during pregnancy, as shown by Mortimer, Wright and Collip.[2]

mentioned the possibility that the active material in such preparations may have originated in the nasal mucosa or may have been present there secondarily.

The possibility that events in the nasal mucosa have an effect on the genital system was mentioned by a number of older writers. Mackenzie [4] in 1884 indicated that not only might nasal irritation and disease result from disorders of the genitourinary tract, but the converse might occur. Fliess [5] in 1897 reported that he controlled, by local treatment of the nasal mucosa, certain forms of dysmenorrhea, and to some extent labor pains. However, if hormones are the intermediary of the nasogenital relationship, then a reciprocal relation would not seem possible. The gonadal hormones might affect the nasal mucosa, either specifically or through some generalized, systemic effect, but one can hardly see how, conversely, a change in the nasal mucosa would affect the vagina or the ovaries, that is, if hormones are the mechanism, as is increasingly apparent. It is

3. Mortimer, H.: Laryngoscope **50**:349 (April) 1940.

4. Mackenzie, J. N.: Am. J. M. Sc. **87**:360, 1884.

5. Fliess, W.: Die Beziehungen zwischen Nase und weiblichen Geschlechtsorganen in ihrer biologischen Bedeutung dargestellt, Leipzig, Franz Deuticke, 1897.

significant that recent studies on treatment of atrophic rhinitis [6] with estrogen have not shown that responses in the sex organs were secondary to the nasal treatments (the dose of estrogen, of course, being well below an amount which would have a direct hormonal effect on the gonads).

Recent literature is significantly much more concerned with the influence which the gonads have on the nose, or at least with the effect on the nasal mucosa of general systemic conditions which are closely related to conditions in the sex organs. Ozena (or atrophic rhinitis), for instance, has received much attention, and in clinical literature its cause is often linked with conditions which suggest a low level of estrogen in the body.

RELATION OF ATROPHIC RHINITIS TO MINIMUM PRODUCTION OF ESTROGEN

In cases of ozena, the approximate period of menstruation has been reported as the most severe for the patient.[7] The improvement in ozena during pregnancy, on the other hand, indicates strongly that in cases in which this occurs there is some deficiency of function during the nonpregnant state. The successful use of estrogen,[9] now a common treatment for ozena, suggests that this deficiency is in the follicular hormone.

Work of Mortimer, Wright and Collip [2] on Estrogen Therapy.—The outstanding contribution in the whole field is the series of experiments by Mortimer, Wright and Collip. First these investigators treated 2 patients suffering from atrophic rhinitis with estrogen and compared their improvement with the condition of 2 controls treated with inactive oil. Later a series of 31 women and 7 men showed highly satisfactory results.

Blaisdell's [1] Work on Estrogen Therapy.—In another notable contribution, that by Blaisdell in August 1937, the treatment of atrophic rhinitis with estrogen was reported, the substance being used at first "merely empirically, [with the hope of obtaining] results similar to those of Davis and Lewis in their treatment of atrophic or senile vaginitis with estrin." The latter authors pointed out the similarity in pathologic changes in atrophic vaginitis and in atrophic rhinitis. Of Blaisdell's 58 normal patients (37 women), the condition of all but 6 was improved. He stated: "In a few cases the improvement has been nothing short of spectacular. In my experience I have never encountered a more grateful and appreciative group of patients." [1]

Negative Reports on Estrogen Therapy.—Blaisdell mentioned, however, the results of Hoople and Rowe, who in 1927, in a review of 80 cases of atrophic rhinitis, concluded that there was no evidence that the endocrine glands have a causal relation to this disease.[1]

Another negative report is that of Strachan,[6] who, with Dr. Wishart, treated 8 children suffering from atrophic rhinitis with a nasal spray of 0.25 cc. of a preparation of an estrogen in oil for three to eight months. The patients were instructed to cleanse their noses at home with Seiler's solution (a solution containing sodium bicarbonate, sodium borate and sodium chloride, with small amounts of eucalyptol, thymol and methyl salicylate). The immediate results

6. Strachan, J. G.: Canad. M. A. J. **44**:158 (Feb.) 1941. Mortimer, Wright and Collip.[2] Blaisdell.[1]

7. Endriss, G.: Die bisherigen Beobachtungen von physiologischen und pathologischen Beziehungen der oberen Luftwege zu den Sexualorganen, Würzburg, Becker, 1892.

8. Endriss.[7] Blaisdell.[1]

9. Mortimer, Wright and Collip.[2] Blaisdell.[1]

were excellent. But a check-up one and a half years later revealed that rhinitis was still present, the disease being alleviated only by constant cleansing with Seiler's solution. They concluded that estrogen therapy was not effective.

It should be mentioned that there is the possibility that atrophic rhinitis is due either to an actual estrogen deficiency or to a relative loss on the part of the mucosal cells of the concha of their specific response to the normal quantities of estrogen in the blood, possibly as a result of phylogenetic development. This may have come about in much the same way that man has almost completely lost the capacity for the "skin" response to circulating "sex hormone," which is so strikingly retained in the perineum, nipple, face and nose in certain modern primates.

> That this capacity, however, is not entirely lost is shown by the reaction which takes place when the hormone is allowed to exert its specific influence locally in concentrations which, while slight in terms of absolute dosage, are relatively high at the site of absorption.[2]

Evaluation of Conflicting Results.—Like other glandular therapy, however, the use of estrogen would be justified even if it were merely palliative, rather than curative, as Blaisdell [1] suggested:

> I do not believe that estrin therapy is a "cure" for atrophic rhinitis, in the sense that we cure other diseases. It may be that we can control the disease better with estrin than with other forms of treatment, just as insulin controls diabetes but does not cure it.

Incidence of Atrophic Rhinitis According to Sex.—Further weight is added to the estrogen theory of the causation of atrophic rhinitis by a consideration of the incidence of the disease as related to sex. More women than men complain of atrophic rhinitis. It is interesting to note, as Blaisdell pointed out, that the greatest amounts of estrogen are found in the urine of male animals, the substance being prepared from the testicles of stallions, since they contain over five hundred times as much estrogen as the ovary of the sexually mature female! Indeed, estrogen has been called by Zondek a decomposition product of androgen. Butenandt even expressed the opinion that both substances arise through progressive decomposition of the sterols.[1] Such an abundance of estrogen products in the male might be considered a contributory cause of the apparent disparity in the incidence of atrophic rhinitis in males and in females.

Incidence of Atrophic Rhinitis in Relation to Dyspituitarism.—Seifert,[10] in his outstanding critical study of the subject up to 1912, mentioned the pituitary gland. He expressed the opinion that its enlargement during pregnancy might possibly have an effect on the nose, especially since in acromegaly the nose as a whole is enlarged. He was more impressed by the possibility of an ovarian influence, but concluded: "It is too early and too risky to explain the nasogenital relationship by the effects of hormones."

Mortimer, Wright and Collip [2] stated that they first became interested in the problem by observing a family in which nasal disease was linked with dyspituitarism and both conditions appeared to have a hereditary basis. Later, they made cranial roentgenograms of 69 patients with atrophic rhinitis and found degrees of cranial dysplasia in 80 per cent of them. They expressed the opinion that there is a two-fold possibility. The pituitary may exercise a direct effect on the nasal skeleton, possibly resulting in the well known saddle nose, an abnormally wide nasal skeleton which predisposes to atrophic rhinitis. Or the dyspituitarism may produce hypogonadism, which may affect the nasal mucosa. Or the pituitary may work in both these ways to bring about the disease.

---

10. Seifert, E.: Ztschr. f. Laryng., Rhin. **5**:431, 1912.

Mortimer, Wright and Collip [2] compiled statistics to show that women tend to suffer from dyspituitarism more often than men. And, as has been shown, atrophic rhinitis is commonly regarded as being more prevalent in women. At any rate, after a conscientious investigation and a review of the literature, they came to the conclusion that this disease gives evidence of a constitutional background associated with dyspituitarism, which often takes the form of hypofunction of the pituitary during some part of the growth period. Cases of atrophic rhinitis with primary amenorrhea might seem to implicate the pituitary as a common link, they suggested.

Menstrual abnormalities were also observed by Blaisdell to be frequent in patients with atrophic rhinitis. Another interesting bit of evidence relating this condition of the nose with disturbances in the genital system is the frequent mention in the literature of olfactory hallucinations in early pregnancy and during the menopause.[2]

CONDITIONS RELATED TO MAXIMUM PRODUCTION OR RELEASE OF ESTROGEN

If such conditions as have already been discussed can be associated with periods when the level of estrogen is presumably low, then it seems logical that the opposite effects might be noted during times when the estrogen level, either by production or release, is regarded as being at the maximum, presumably during pregnancy and in the latter part of the menstrual cycle.

Vasomotor Rhinitis During Pregnancy.—In my clinical experience I find that when the estrogen level is high there is a definite tendency toward hyperemia and swelling of the mucous membranes. In fact, my attention was first attracted to this problem by patients with vasomotor rhinitis and associated allergic states who are referred by obstetricians. A certain number of women suffer to some extent during the last two months of pregnancy, but this is taken largely as a matter of course.

Of the patients referred to me during pregnancy, I have studied 8 who gave a negative history of any such trouble during the nonpregnant state. One of my patients had similar disturbances during three previous pregnancies; 2 patients, during two previous pregnancies, and 2 patients, during one previous pregnancy. The majority of these patients were very uncomfortable, and all the usual forms of palliative therapy for allergy seemed to be of little value. Only 1 patient had a secondary sinus infection. The periods of onset varied from the third to the seventh month and in all cases persisted to term. All of the patients showed either cure or definite improvement within one week post partum. Several were relieved within twenty-four hours after delivery.

The appearance of the mucous membranes was fairly constant from one patient to another. When the patient was first seen with these complaints, the nasal changes were mostly those of engorgement and swelling and increased redness of the mucous membranes. As the pregnancy progressed the symptoms became more pronounced, and the majority of the patients showed gradually increasing swelling and redness, and some, marked edema of the mucous membranes.

Eosinophil counts of the nasal secretions were made on 5 patients. Two women showed 2 per cent; 1, 4 per cent; 1, 5 per cent, and 1, 6 per cent. The basal metabolic rates were determined for 3 patients and were found to be within normal limits for pregnancy.

In addition to the aforementioned 8 patients, another series of 12 women showed some previous history of allergic disturbances, such as seasonal hay fever or perennial nasal allergy. These 12 women all showed a pronounced increase in their

nasal symptoms, the reaction being more that of acute hay fever. For most of these patients the nasal secretions showed an eosinophil count above 4 per cent. The mucous membranes were intumescent and pale, with a bluish background.

Typical Case of Vasomotor Rhinitis During Pregnancy.—The following case history is taken from the first group of patients.

Mrs. W. J. O., aged 31, was first seen by members of the obstetrics department on Aug. 4, 1939. Her delivery was expected on March 2, 1940. The history was negative for nasal disturbances except that during one previous pregnancy she had had considerable nasal blocking. The results of complete physical examination at this time were essentially normal. Inspection of the nose showed slight deviation of the septum to the right, reddening of the mucous membrane and slight enlargement of the turbinates. The tonsils had been removed.

Data on Two Series of Women with Vasomotor Rhinitis During Pregnancy

Case No.	Name	Age, Yr.	Nasal Disturbance During Previous Pregnancies	Abnormalities of Nose	Eosinophil Count, %	Degree of Nasal Blocking: First Examination	Degree of Nasal Blocking: Mid-pregnancy	Degree of Nasal Blocking: Term	Return to Approximately Normal Post Partum
				Group A *					
1	W. J. O.	31	Yes-2	Septum deviated slightly to right	6	1+ (4 mo.)	3+	4+	48 hr.
2	T. R. J.	28	No	None	2	3+ (3 mo.)	4+	4+	72 hr.
3	J. L. M.	24	Yes-1	Septum deviated moderately to right	..	1+ (3 mo.)	2+	3+	5 days
4	M. R.	33	Yes-3	Middle turbinate slightly polypoid	4	2+ (2½ mo.)	3+	4+	4 days
5	H. S. B.	28	Yes-2	None	2	3+ (3 mo.)	3+	3+	72 hr.
6	R. E.	24	No	None	..	1+ (3 mo.)	3+	4+	48 hr.
7	S. O. J.	27	Yes-1	Septum placed high to left	..	2+ (4 mo.)	3+	3+	72 hr.
8	M. E. M.	20	No	None	5	1+ (3 mo.)	3+	4+	4 days
				Group B †					
1	H. S.	21	No	None	6	1+ (2 mo.)	3+	4+	1 wk.
2	A. B.	24	No	Septum deviated to right	..	2+ (3 mo.)	3+	3+	10 days
3	C. O. K.	29	Yes-2	Middle meatus polypoid	4	3+ (3 mo.)	4+	4+	4 days
4	A. C. M.	26	Yes-1	None	2	3+ (3 mo.)	4+	4+	3 days
5	P. O. B.	22	No	Septum deviated moderately to left	..	4+ (3 mo.)	4+	4+	6 days
6	S. A.	24	Yes-2	Middle meatus polypoid	..	2+ (3 mo.)	3+	3+	3 days
7	C. A. A.	33	No	None	..	1+ (3 mo.)	2+	3+	48 hr.
8	R. P.	31	Yes-1	None	..	2+ (3 mo.)	2+	2+	3 days
9	A. J.	27	No	None	12	3+ (3 mo.)	4+	4+	12 days
10	M. M.	24	No	Middle meatus polypoid	..	4+ (3 mo.)	4+	4+	Indefinite
11	M. A. H.	28	No	None	3	2+ (2 mo.)	2+	3+	4 days
12	B. L. M.	23	No	None	7	3+ (3 mo.)	3+	4+	6 days

* Eight patients who had never shown any symptoms of nasal obstruction except during pregnancy.
† Twelve patients who showed marked nasal obstruction during pregnancy, all of whom gave a history of some previous allergic or vasomotor nasal disturbance during nonpregnant periods.

On October 11 the patient was seen by members of the department of laryngology. Her chief complaint was blockage of the nose and a clear watery discharge during the previous week. The eosinophil count of the nasal secretion was 4 per cent. Examination showed that the mucous membranes were swollen. For the most part, they were deep red, but an occasional pale area was seen.

From this time on she was seen at intervals of two weeks. Symptoms of nasal blockage became gradually more pronounced. On Jan. 10, 1940 the eosinophil count was 6 per cent.

She was delivered of a normal boy on March 9. Her nasal symptoms commenced to clear within forty-eight hours post partum and had completely disappeared by the fourth day.

She again became pregnant on Sept. 2, 1940 and by Jan. 6, 1941 she began once more to have nasal involvement, which followed the same course as before. Two basal metabolism tests gave readings of —3 and —5 per cent. The calcium of the blood measured 12 mg. per hundred cubic centimeters and the phosphorus 5 mg. Complete tests for allergy gave chiefly negative reactions. Tests for foods and inhalants gave negative responses. Of the grasses, redtop, vernal koeleria and cultivated oats gave a plus-minus reaction.

None of the usual palliative measures afforded relief. After delivery her condition once more cleared up rapidly.

Infection of the Maxillary Sinus During Pregnancy.—Closely related to the two series of patients with vasomotor rhinitis is another group of 9 patients whom I treated for infection of the maxillary sinus during pregnancy. In each of these patients the condition was definitely more stubborn than usual in resisting ordinary therapy. In 4 of the patients, in spite of antral lavage two months before delivery, the infection showed no signs of clearing. Yet post partum the condition cleared up with few irrigations. The disturbance may be based on swelling of all the mucous membranes, including the lining of the antrum. All the membranes of the nose were much more swollen than is usual.

Increase in Estrogen During Pregnancy.—A predisposition to this condition parallels closely the production of estrogen. As Blaisdell [1] stated:

> As soon as pregnancy occurs and the fertilized ovum has come into contact with the maternal circulation, massive production of estrin sets in rather acutely. . . . The amount steadily increases throughout pregnancy, so that in the first trimester of pregnancy one litre of urine contains about 2,000 to 3,000 mouse units; towards the middle of pregnancy, 10,000 mouse units, and just before parturition, 50,000 units.

Epistaxis During Pregnancy.—Nosebleed, another condition related to hyperemia, has also been reported by a number of workers as frequent [8] and as present in 25 per cent,[11] in 22 per cent [12] and in 50 per cent of cases.[2]

Premenstrual Colds.—Premenstrual colds present another interesting relationship. As Mortimer, Wright and Collip [2] pointed out when they were asked whether estrogen treatment would be helpful for this difficulty, premenstrual colds tend to indicate overproduction, rather than underproduction, of estrogen at this period, and estrogen therapy would therefore be contraindicated.

PHYSIOLOGIC RELATIONSHIP

These more or less pathologic relations tend to indicate the possibility of a normal, physiologic nasogenital relationship. More accurately, perhaps, this should be referred to as the effect on the nasal and vaginal mucous membranes of a generalized response to hormonal production levels. There might be expected in the nose some reflection of physiologic events, such as menstruation and pregnancy.

Menstruation.—The relation to menstruation has been investigated in monkeys.[2] A colony of 28 animals were examined objectively, with striking results. In normal females, both adult and immature, peaks of nasal activity (shown by redness and/or swelling of the mucosa conchae) occurred periodically at intervals of about twenty-eight days and were synchronous with the changes known to occur in the nipple, sex skin, face and back. The peaks were chiefly premenstrual. The authors even found that such peaks of nasal activity were 8 per cent more constant than menstruation itself in indicating the sex cycle. The activity was most evident in the autumn, when conception is most likely to occur.

This parallels Zondek's conclusions regarding production of estrogen in man, namely, that it falls to a minimum after each menstruation but never ceases, and that it attains its maximum in the premenstrual stage. Excretion in the postmenstrual phase is about 50 mouse units, and in the intermenstrual phase about 300 mouse units, per liter of urine.[1]

Imitation with Estrogen.—Mortimer, Wright and Collip [2] found that the administration of estrogenic substances produces an imitation of the premenstrual condition, with respect to the changes in the nose, nipple, sex skin, face and back, in both

11. Kindermann, V., and Eichbaum, F.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **89**:230 (Nov. 3) 1936; **86**:284 (Nov. 13) 1935.
12. Abelin, I.: Biochem. Ztschr. **286**:160, 1936.

male and female animals, adult and immature, intact and castrate. In other words, the tendency of estrogen treatment to produce hyperemia, and thereby to relieve atrophic rhinitis, has its physiologic counterpart in the premenstrual period of the sex cycle. Moreover, this period of the sex cycle may produce the opposite type of difficulty, namely, the premenstrual cold.

Imitation with Testosterone.—The close relation between androgen and estrogen, which was previously mentioned, led another group of workers to observe the effects of testosterone propionate on monkeys and on 8 human subjects. They noted the same type of changes as those reported for estrogen therapy, i. e., congestion, swelling and edema.[1]

Contraindications to the Use of Testosterone and Estrogen.—However, the use of androgens in cases of ozena is contraindicated because of their direct masculinizing effect. After he had considered the experimental reports of Burrows and Rush, Blaisdell [1] stated that the use of estrogen in treatment of male patients is not contraindicated. Nor is the use of estrogen contraindicated from the standpoint of a carcinogenic action, as such an effect is unlikely. Blaisdell [1] found the only contraindication in occasional cases of severe acute sinusitis, although headaches, nausea and hypersensitivity to sesame oil were encountered.

Pregnancy.—Observations on human subjects during pregnancy indicated another parallelism. From a preliminary study of 60 pregnant women during irregular periods, Mortimer, Wright and Collip drew up a composite picture.

> In the second month of pregnancy the nose is entirely normal. By the end of the 3rd month there is a tendency for a slight increase both in colour and activity, still, however, within normal limits. By the end of the 4th month the top limit of normal coloration has been reached, but swelling is not a marked feature. By the end of the 5th month the colour is slightly above normal limits, while at the end of the 6th, it is somewhat higher, and at the 7th it is unmistakably higher. It is not, however, until the 8th month that all the cases, with one exception, showed an almost maximal redness, which is sustained until delivery. Swelling is not as a rule a marked feature or one of which one can be very sure, until the end of the 7th month, and thereafter it is usually a well marked feature.[2]

All of which corroborates the well known observations of many early workers.[13] As Mortimer, Wright and Collip pointed out, the graph which they built on these data corresponds closely with the known excretion of estrogen during pregnancy, which was previously described in a citation from Zondek.

Summary of Relationships.—From many standpoints, therefore, and in several different ways, there is a correlation between the nose and the genital system. That estrogen plays an important part in this systemic condition can hardly be doubted, although various predisposing factors may modify to a considerable extent the effect of the estrogen present. The pituitary may exert its influence either directly on the nose or indirectly by its influence on the production of estrogen.

> These are facts of paramount importance to the rhinologist in his conception of the etiology of nasal diseases. . . . The possible influence of the ovary in such conditions as hypertrophic rhinitis and atrophic rhinitis cannot be disregarded. Even in such questions as individual susceptibility or resistance to the common cold, as well as in a tendency to the manifestation of allergic states, appearing in a person who has never before shown such manifestations, there may be an indication of the gradual effect of hormone disturbance, in the sexual sphere, making itself felt in local nasal changes.[2]

In the broader aspect, the effect of either ovarian or testicular secretion may be a definite factor in vasomotor rhinitis, in cases aside from those of pregnancy.

---

13. Zacharias, P.: Med. Klin. **3**:58, 1907. Freund, H. W.: Monatschr. f. Geburtsh. u. Gynäk. **20**:210, 1904. Fliess.[5]

ANTIHORMONES AND ALLERGY AS RELATED TO ESTROGEN AND VASOMOTOR RHINITIS

From the foregoing discussion, it appears that an overabundance of estrogen in the body may be accompanied by vasomotor conditions closely resembling the allergies. There arises the question whether the vasomotor condition might be associated with an allergic reaction to estrogen or to some undefined impurities related to its production.

The question whether or not hormones can produce allergic responses is one which seems, unfortunately, to have become a bit confused in the literature. According to Collip and others, the antigenicity of hormones, that is, their ability to produce antibodies in the blood and cause allergy, is an entirely different concept from that of the ability of hormones to produce antihormones.[11]

Antihormones.—It is now a well established fact, first reported by Abelin [12] in 1928 and extensively investigated by Collip,[14] that prolonged treatment with a glandular preparation results in resistance of the body to the specific glandular principle. Collip explained this with his theory of antihormones, which he concluded were not the result of an antigen-antibody response. Unlike the antibodies produced in serologic reactions, these antihormones cannot be transmitted from immune to untreated animals, as has been discovered with respect to the antihormone against estrin [15] and against thyroxin.[16] Antihormones have been reported for epinephrine and the thyrotropic and gonadotropic substances.[17] The concept of antihormones suggests a possibility as to the immediate causation of the pathologic reactions of the nose associated with estrogen. The level of antihormones against estrogen in the body might modify to a considerable extent the effective level of the quantity of estrogen present.

Allergy to Hormones.—Entirely separate from the question of antihormones is that of allergy as related to hormones, although in some cases antibodies seem to develop in an animal at about the same time as antihormones, in response to a series of administrations of glandular preparations.[16] Thus, Bauer and associates found that they were able to produce serologic reactions against thyroxin at about the same time that biologic resistance to thyroxin developed in their animals, although this resistance was not produced by the antibodies. (Presumably it was a result of antihormonization.) Bauer and his associates also observed positive complement fixation reactions in animals and human subjects showing various signs of hyperthyroidism. It should be mentioned that he found that these serologic reactions to thyroxin were not specific for thyroxin, but were directed against the entire phenol group: diidotyrosine, epinephrine and, in most cases, insulin.

However, thyroxin and its phenol ring associates are proteins, and antigenicity might be expected. Fatty substances, such as estrogen and androgen, do not have this property of antigenicity.[18] A number of workers have obtained comple-

14. Collip, J. B.: Ann. Int. Med. **8**:10 (July) 1934; **9**:150 (Aug.) 1935.

15. D'Amour, F. E.; Dumont, C., and Gustavson, R. G.: Proc. Soc. Exper. Biol. & Med. **32**:192-194 (Oct.) 1934.

16. Bauer, J.; Kunewälder, E., and Schächter, F.: Wien. klin. Wchnschr. **49**:39 (Jan. 10) 1936; **50**:83 (Jan. 22) 1937. Bauer, J., and Kunewälder, E.: ibid. **50**:399 (March 26) 1937.

17. Bauer and associates.[16] Kindermann and Eichbaum.[11]

18. Schmidt, C., and Allen, F. W.: Fundamentals of Biochemistry with Laboratory Experiments, New York, McGraw-Hill Book Company, 1938.

ment fixation or precipitin reactions against the gonadotropic substances,[19] but all these authors except Ehrlich admitted that the serologic antibody reaction is or might not be against the principle itself but against some impurity. Finch's work is closely related to our own study.

Allergy to Progestin.—Finch [20] reported that an allergic response to progestin is a factor in nausea during pregnancy. His results were obtained with the complete corpus luteum secretion—progestin—and could not be duplicated by the use of the purified progesterone, a result suggesting that some factor other than the hormone itself is the agent. He gave intradermal injections of extracts of corpus luteum to pregnant patients suffering from nausea, and reactions developed in direct proportion to the degree of nausea present. Thirty patients treated with desensitizing doses were relieved to varying extents.

The output of the corpus luteum seems to parallel the usual duration of nausea, increasing during the first few months and then subsiding gradually.

Hirst [21] originally connected nausea with function of the corpus luteum, although he expressed the belief that the normal woman, accustomed during her menstrual life to secretion of corpus luteum, suffers nausea in pregnancy as the result of being suddenly deprived of the hormone. As Finch [20] pointed out, the relief which Hirst obtained for his patients may have been due to a sensitizing process rather than to providing the patients with a secretion in which they were deficient.

Finch expressed the belief that nauseated patients either have inherited directly a sensitivity to progestin, or, more probably, have inherited the allergic tendency and become sensitive to their own corpus luteum secretion during their menstrual cycles or during a previous pregnancy. His observations were as follows: 1. A high percentage of patients with nausea had other diseases of an allergic nature or gave a family history of such diseases as hay fever or asthma. 2. Patients without nausea reacted negatively to the cutaneous test. 3. Patients with nausea reacted in proportion to the amount of nausea. 4. Patients with nausea were desensitized and nausea was relieved. 5. The positive cutaneous reaction was obtained before as well as during pregnancy.

Possible Etiologic Factors in Vasomotor Rhinitis During Pregnancy.—In the problem of vasomotor rhinitis, such an etiologic factor as that just discussed seems ruled out. Estrogen, being a fatty substance, could not as a pure hormone have real antigenicity. Any antigens would have to be in the form of undefined impurities (presumably protein) related to the production of estrogen. Yet it seems to be the estrogen itself which, when injected into monkeys, duplicates the premenstrual, and to a certain extent the pregnant, nasal mucosa. Likewise, it seems to be the estrogen itself, rather than the impurities, which is the effective treatment for the reverse nasal condition of atrophic rhinitis. Antihormones, or rather a lack of antihormones to estrogen, might be a contributory causal factor.

Dyspituitarism may be involved, as it seems to be in cases of atrophic rhinitis. It may be possible to trace the ovarian factor back to the associated pituitary fraction. While hypopituitarism was found to be more frequent in women than hyperpituitarism,[2] nevertheless a tendency to hyperpituitarism, in addition to the

19. Ehrlich, H.: Wien. klin. Wchnschr. **47**:1323 (Nov. 2) 1934. Bachman, C.: Proc. Soc. Exper. Biol. & Med. **32**:851 (March) 1935. Brandt, R., and Goldhammer, H.: Ztschr. f. Immunitätsforsch. u. exper. Therap. **88**:79 (May 30) 1936. Twombly, G. H.: Endocrinology **20**:311 (May) 1936. Kindermann and Eichbaum.[11]

20. Finch, J. W.: Am. J. Obst. & Gynec. **40**:1029 (Dec.) 1940; Etiology of Nausea and Vomiting of Pregnancy, J. A. M. A. **111**:1368 (Oct. 8) 1938.

21. Hirst, B. C.: A Textbook of Obstetrics, ed. 8, Philadelphia, W. B. Saunders Company, 1918.

great normal production of estrogen during pregnancy, might tend to bring about the symptoms. The acromegaly of pregnancy may in some way predispose the nasal structure to vasomotor rhinitis.

Further study is indicated, and I hope in a new series of cases to obtain data on several of these points in question, especially in regard to the determination of estrogen.

SUMMARY

Vasomotor rhinitis and associated conditions have a definite incidence during pregnancy.

This paper reports the cases of 8 pregnant women who experienced a severe degree of nasal blocking and congestion during their pregnancy, while in the nonpregnant state none of these women had ever experienced any such trouble. Twelve other pregnant women experienced severe nasal blockage, but these patients had a history of previous transient attacks of hay fever and nasal blocking in the nonpregnant state. In 9 patients with purulent sinusitis the infection was more protracted and the mucous membrane more swollen than is usually encountered. They showed no response to the usual therapy, but the condition cleared quickly after delivery in all instances.

From both a theoretic and a clinical standpoint, allergy did not seem to be the predominant etiologic factor.

The incidence of vasomotor rhinitis during pregnancy appears to parallel and to be caused by the amount of estrogen produced in the body.

Typically the condition disappears spontaneously after delivery.

23 Second Avenue.

UNILATERAL VASOMOTOR RHINITIS DUE TO INTERFERENCE WITH THE CERVICAL SYMPATHETIC SYSTEM

EDMUND PRINCE FOWLER Jr., M.D.
NEW YORK

It was my fortune to be assigned the patient to be reported on by Dr. Charles Depping, who, even with the necessarily cursory examination of a busy chief of clinic and distribution officer, recognized the possible importance of the case in the search for a more complete understanding of allergic disease.

REPORT OF A CASE

History.—V. H., a widowed typist aged 34, presented herself at the clinic of the Manhattan Eye, Ear and Throat Hospital in November 1940. Her chief complaint was stoppage of the left side of the nose of fifteen months' duration and a watery secretion, with much sneezing, of three months' duration. There was no history of allergy in the family. She herself had had no symptoms suggestive of allergy before her present illness. Rarely had she had colds or sore throat and never ocular symptoms.

In February 1939 a small lump which had been on the left wrist for twenty years without enlarging was diagnosed in another hospital as a cyst. It gave no symptoms except a slight sensation of electric shock on pressure. On exposure and removal the tumor proved to be a neurofibroma of the median nerve. After the operation there was extreme pain in the hand. The diagnosis was causalgia of the median nerve. Neurolysis was performed by another surgeon but gave no relief. Infra-red radiation, whirlpool baths and casts for the arm were all ineffectual. Injections of alcohol afforded only temporary relief of the pain. A "secondary tumor" developed at the old site, which could not be touched because of its tenderness. Because of unbearable pain, she consulted a neurosurgeon, who removed the left stellate ganglion in August 1939. The patient claimed that on her recovering from the ether there was no alleviation of the pain in the wrist. Further, there were drooping of the left eyelid and constriction of the pupil (Horner syndrome), pain in the left eye and stuffiness in the left nostril. Shortly afterward she began to complain of difficulty in breathing, a feeling of tightness around the heart and absence of perspiration over the left hand. With the beginning of cold weather in September 1940, there appeared sneezing spells, lasting thirty minutes; the left side of the nose became constantly stopped, and the watery secretion from it was so uncontrolled that it interfered with her work. It dropped on her shorthand pad and spotted the letters on her desk.

From the Manhattan Eye, Ear and Throat Hospital; Clinic of Dr. Joseph D. Kelley.

Prepared for presentation at the Forty-Eighth Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Atlantic City, N. J., June 3, 1942.

Examination.—There was a typical Horner syndrome on the left side, with pronounced drooping of the upper lid and constricted pupil. The skin of the left side of the face and the left hand appeared dry and lifeless. There was slight atrophy of the subcutaneous tissues of the hand. A well healed linear scar was present just above the middle of the left clavicle.

The nasal septum was moderately deviated to the left, and the passage was decidedly narrowed on this side because of the pale, edematous mucous membrane over both the septum and the turbinates. In color and appearance the mucous membrane on the left side was typical of that often seen in cases of uncomplicated allergic rhinitis. On the right side the passage seemed normal in every way. Smears of the secretion showed only a few epithelial cells on the normal side, but large numbers of eosinophils on the affected side. Cutaneous tests for the common inhalants—ragweed, timothy, June grass, red top, orris, dog and cat hair and horse dander—and for the foods wheat, milk, eggs, chocolate and oranges gave negative results. The hemoglobin content was normal, as were the white cell and the differential count. The Kahn reaction was negative, and roentgenograms of the sinuses revealed nothing abnormal.

Course.—Shrinkage of the enlarged inferior turbinate with a bipolar diathermy needle improved the airway slightly, but not enough for comfort. Nose drops of ephedrine and epinephrine seemed to make the symptoms worse. Shrinkage with cocaine was slow and did not last long. Administration of atropine sulfate, $\frac{1}{250}$ grain (0.2 mg.) every two or three hours by mouth, stopped the copious secretion and produced satisfactory symptomatic relief, but it could not be continued long because of the general reactions and the fear of atropine poisoning. Submucous resection of the deviated septum was performed April 2, 1941. The inner surfaces of both flaps were scarified in the hope that some of the sympathetic fibers from the normal side would grow across. The operation gave only slight relief. The patient was then referred to Dr. Clarence Hare, who advised no further operative intervention on the wrist and recommended the oral use of syntropan.[1] At first 100 mg. was used three or four times a day, with notable relief about ten minutes after the capsule was taken, and without unpleasant effects on the eyes or the general well-being. The patient has now been taking syntropan since May 7, 1941, with the exception of June, July and August 1941, at which time the nose showed marked improvement with the heat and the dry weather and there was only occasional necessity for taking the drug. Such periods seemed to occur during changes in weather or after exposure to a draught. With the arrival of cold weather in the fall of 1941, the nasal symptoms returned and continued throughout the winter. Even when syntropan was taken, the blockage of the nose was seldom relieved. However, the sneezing and watery discharge were controlled by the drug. Beginning about May 7, 1942, the symptoms have been regressing again. It is now necessary to take only 1 (100 mg.), or occasionally 2, capsules per day. The patient has noticed that when she is very warm and the room temperature is constant the nose is "very good." If the window is opened or any change in the air current appears, sneezing immediately occurs, with dripping from the nose, congestion and itching of the left nostril and a feeling of fulness in the head. The itching of the left nostril persists after the other symptoms cease.

1. Syntropan, a synthetic product manufactured by Hoffmann-La Roche, Inc., is the phosphate salt of the dl-tropic acid ester of 3-diethylamino-2,2-dimethylpropanol.

COMMENT

Since seeing this patient, I have examined the noses of 4 other patients with Horner's syndrome. One with a condition of ten years' duration claims to have had blockage and watery secretion from one side of his nose after resection of the cervical sympathetic fibers for peripheral vascular disease.' He had complete relief after a submucous resection. The mucous membrane on both sides of his nose appeared normal, in spite of persistence of the Horner syndrome. Another patient, a woman, had traumatic severance of the cervical portion of the trunk one year before examination, with faint residual ocular symptoms. The nose appeared normal, and she could remember no symptoms. Two other patients with a preganglionic Horner syndrome, one with syringobulbia and the other with an unlocalized intracranial lesion, showed no involvement of the nasal mucosa that I could see and complained of no symptoms suggestive of vasomotor rhinitis. I heard of another patient, an elderly woman in a private hospital, from a physician observer who knew I was interested, but I was not permitted to examine her. This woman's chief complaint after injection of alcohol into the left stellate ganglion for pain of coronary origin was said to be blockage of the left side of the nose and copious secretion therefrom.

The cooperation of neurologists and surgeons operating on the peripheral neurovascular apparatus is necessary for the further study of this problem. Many cases of the Horner syndrome representing all etiologic factors should be collected, and the nasal passages should be examined by rhinologists. Careful note should be made as to the portion of the sympathetic nervous system involved in each case. It is possible that the vasomotor changes in the nose occur or are only permanent, with a lesion of the stellate ganglion. By collecting all types of Horner's syndrome one may localize the control station for vasomotor rhinitis in human beings.

It has been known for a long time that hay fever, asthma and other allergic diseases can be controlled to a certain extent by drugs which stimulate the sympathetic nervous system or inhibit the parasympathetic system. The case reported here, because of the specific character of the operative lesion, suggests that experimental surgery might uncover the fundamental mechanism of vasomotor rhinitis. If one can produce experimental vasomotor rhinitis at will, one should be a long way toward understanding allergic diseases more thoroughly. Animal surgery may localize the part of the sympathetic or the parasympathetic nervous system which needs to be attacked for prevention or cure.

140 East Fifty-Fourth Street.

PAREDRINE HYDROBROMIDE–SULFATHIAZOLE THERAPY OF INFECTIONS OF UPPER RESPIRATORY TRACT

LOUIS D. SULMAN, M.D.
PHILADELPHIA

Before the introduction of therapy with the sulfonamide compounds there were no satisfactory inhibitors of bacterial growth in the nasal cavity. Silver salts came nearest to solving the problem, but they precipitated and thus clogged the cilia. Argyria occasionally resulted.

The bacteria most frequently observed with infections of the upper respiratory tract, according to Ashley and Frick,[1] occur in this order: Staphylococcus aureus, Micrococcus catarrhalis, Staphylococcus albus, the beta and alpha hemolytic streptococci, Streptococcus foecalis, the influenza bacillus, Bacillus mesentericus and the pneumococcus. Any of these organisms can be responsible for a secondary infection after the initiation of a cold. Sinal infections are usually attributed to the streptococcus, the pneumococcus and the staphylococcus.

Many investigations have shown that sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) is the most effective of the sulfonamide compounds so far studied against the majority of the bacteria present in the nasal cavity. For example, Osgood,[2] in his studies on cultures of the human bone marrow, found that the action of sulfathiazole was equal or superior to that of sulfanilamide against the beta hemolytic streptococcus and to that of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) against the pneumococcus. Sulfathiazole was greatly superior to sulfanilamide against the alpha hemolytic streptococcus and was effective against Staph. aureus and Staph. albus, whereas neither sulfanilamide nor sulfapyridine was very effective. Schmidt and associates[3] reported that sulfathiazole was more effective than sulfapyrazine (2-[paraaminobenzenesulfonamido]-pyrazine), sulfapyridine or sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine) against Pneumococcus types 1, 2 and 3 in beef broth. Rammelkamp[4] showed that sulfathiazole had a bactericidal as well as a bacteriostatic action on Staph. aureus under conditions in which sulfanilamide was neither bactericidal nor bacteriostatic. Poston[5] reported that sulfathiazole inhibited more strains of Staph. aureus than any of the other sulfonamide drugs studied.

Sulfathiazole administered intravenously or by mouth in sufficient quantities to be effective against masses of bacteria such as can collect in the sinuses and eustachian tubes may have toxic effects. It thus becomes necessary for the patient to be seen daily by his physician in order that any possible toxic effects be observed and

1. Ashley, B. J., and Frick, W. V.: A Bacteriologic and Cytologic Study of the Maxillary Antrum in Children, with a Clinical Study of Eighty-Four Cases, Ann. Otol., Rhin. & Laryng. **39**:605-630 (June) 1930.

2. Osgood, E. E.: Chemotherapy, Arch. Otolaryng. **34**:961-968 (June) 1941.

3. Schmidt, L. H.; Ruegsegger, J. M.; Sesler, C. L., and Hamburger, M., Jr.: Sulfapyrazine (2-Sulfanilamide-Pyrazine): Its Antipneumococcal Activity as Compared with That of Sulfapyridine, Sulfathiazole and Sulfadiazine, J. Pharmacol. & Exper. Therap. **73**:468-473 (Dec.) 1941.

4. Rammelkamp, C. H.: Sulfathiazole, Indust. Med. **10**:131-136 (April) 1941.

5. Poston, M. A.: Comparison of in Vitro Effectiveness of Various Sulfonamides on One Hundred and Ten Strains of Staphylococcus Aureus, J. Bact. **43**:74 (Jan.) 1942.

the treatment adjusted accordingly. Because sulfathiazole is only slightly soluble and therefore only slowly absorbed into the system, the logical procedure is to bring the drug into direct contact with the bacteria. Although there are frequent reports on the topical application of sulfathiazole, the literature shows comparatively few references to its use, or even to the use of sulfanilamide, in the nose. Sulfathiazole has been applied as a snuff by Delafield, Straker and Topley,[6] with encouraging results. Connell and Trowbridge[7] insufflated powered sulfanilamide or sulfathiazole into the nasal cavity for chronic rhinitis and sinusitis and in postoperative care. Osgood[2] reported that sulfathiazole has been employed as a heavy suspension either in a sterile saline solution or in a viscid water-soluble medium, but no results were given. He had success with powdered crystalline sulfathiazole in the prophylactic treatment of postoperative wounds in infected areas of the nose, mouth and throat. Marks[8] found a sulfathiazole jelly effective in the treatment of 70 per cent of his patients with chronic maxillary sinusitis.

It has long been thought that the ideal medication of the nose would be one which would both reduce nasal congestion and destroy pathogenic organisms without injury to the cilia. In an earlier study[9] I reported on the rapid and lasting shrinkage of the nasal mucous membrane following the use of paradrine hydrobromide (p-hydroxy-α-methylphenethylamine hydrobromide). A 1 per cent solution of the drug was effective in one to fourteen minutes. Silcox[10] also reported the efficient vasoconstrictor action of paredrine hydrobromide. A combination of paredrine hydrobromide and sulfathiazole would seem to be the ideal medicament. The paredrine component reduces the nasal congestion rapidly and effectively, while the sulfathiazole combats many of the bacteria present. The addition of 5 per cent of microcrystals of sulfathiazole to a 1 per cent aqueous solution of paredrine hydrobromide[11] gives a white, practically odorless, suspension with a p_H of 5.5, a value which falls within the normal p_H range of the nasal secretion in situ, according to Fabricant.[12] Its vasoconstrictor action is about as rapid as that of paredrine hydrobromide alone. The microcrystals form a true suspension, which is not obtained with other forms of sulfathiazole. This suspension does not cake and hence will not clog the cilia. Scheie and Leopold[13] stated that the bacteriostatic properties of microcrystalline sulfathiazole are the same as those of ordinary preparations of the drug. Bacterial invasion of the nasal cavity is not confined to the superficial mucous membranes, but involves the sinuses, the eustachian tubes and the cavities of the middle ear and mastoid. The microcrystals of sulfathiazole are 5 to 10 microns in diameter, small enough to pass

6. Delafield, M. E.; Straker, E., and Topley, W. W. C.: Antiseptic Snuffs, Brit. M. J. **1**:145-150 (Feb. 1) 1941.

7. Connell, E. S., and Trowbridge, B. C.: Local Use of Sulfanilamide and Its Derivatives in the Ear, Nose and Throat, Arch. Otolaryng. **35**:740-744 (May) 1942.

8. Marks, R. F.: Sulfathiazole in the Treatment of Sinusitis, Arch. Otolaryng. **35**:794-798 (May) 1942.

9. Sulman, L. D.: Paredrine in the Treatment of Sinusitis, M. Rec. **150**:27-29 (July 5) 1939.

10. Silcox, L. E.: Rhinitis: A Nasographic Study; Preliminary Report, Arch. Otolaryng. **34**:33-46 (July) 1941.

11. The solution used was paredrine hydrobromide aqueous, which is p-hydroxy-α-methylphenethylamine hydrobromide, 1 per cent, made isotonic with sodium chloride and preserved with merthiolate, 1:100,000. The paredrine-sulfathiazole suspension was supplied by the Smith, Kline & French Laboratories, Philadelphia.

12. Fabricant, N. D.: Significance of the p_H of Nasal Secretions in Situ, Arch. Otolaryng. **34**:297-301 (Aug.) 1941.

13. Scheie, H. G., and Leopold, I. H.: Penetration of Sulfathiazole into the Eye, Arch. Ophth. **27**:997-1004 (May) 1942.

through a no. 26 needle and thus small enough to penetrate the apertures of the structures communicating with the nasal cavity.

Chambers and associates[14] have recently reported that sulfathiazole was superior to sulfanilamide powder for local application in traumatic wounds because of its effectiveness against many types of bacteria. The microcrystals which they used showed less tendency to clump than other forms of sulfathiazole powder tested. They found no evidence of foreign body reaction.

The suspension of sulfathiazole in an aqueous solution of paredrine hydrobromide has none of the caustic effects reported for sodium sulfathiazole. Futch and associates[15] observed that a 5 per cent solution of sodium sulfathiazole, instilled into both nostrils of the rabbit, caused thickening of the nasal mucosa in two days. The cilia became indistinct, and the columnar and basal cells showed vacuolation. Destruction progressed over ten days, and after ten days of rest there was practically no recovery. A 5 per cent solution of sodium sulfathiazole has a p_H of approximately 10. This high degree of alkalinity alone is presumably responsible for the rapid and devastating destruction of the mucosa. A similar caustic action of sodium sulfathiazole has been reported in human subjects by Fletcher.[16]

CLINICAL USE

Over 75 patients were treated with the suspension of sulfathiazole in an aqueous solution of paredrine hydrobromide at Doctors Hospital, Philadelphia, and St. Vincent's Hospital and in private practice. The adults were suffering from acute and subacute rhinitis or from exacerbations of chronic sinusitis, mostly of nonallergic origin. In treatment both in the office and in the hospital the nares were first shrunk with a 1 to 3 per cent solution of paredrine hydrobromide, and suction was applied. Then either the nares were packed with the paredrine-sulfathiazole preparation or drops were instilled. For use in the displacement method of Proetz, the paredrine-sulfathiazole preparation was diluted 1 to 5 with sterile physiologic solution of sodium chloride. The displacement method was used twice a week as long as necessary. In home treatment 2 to 5 drops of the paredrine-sulfathiazole preparation was instilled in each nostril two to four times daily until cure was obtained. Relief of the symptoms was immediate, and cure was prompt when the treatment was given early. Conditions of long standing responded more slowly, but in every instance there were immediate relief and continuous improvement of the condition. The results were so satisfactory that no other therapy was tried with these patients. The data on 7 representative patients are summarized in table 1.

The suspension of sulfathiazole in an aqueous solution of paredrine hydrobromide was applied by the Proetz method in the treatment of a series of infants up to 5 years of age at St. Vincent's Hospital; these patients were suffering from subacute and chronic rhinitis and sinusitis. The method was the same as that previously described. Observations were recorded within twenty-four hours, and additional Proetz treatments were given if necessary, but not more often than

14. Chambers, L. A.; Harris, T. N.; Schumann, F., and Ferguson, L. K.: The Use of Microcrystals of Sulfathiazole in Surgery, J. A. M. A. **119**:324-327 (May 23) 1942.

15. Futch, C. E.; Rosenvold, L. K., and Stewart, C. E.: Caustic Effect of Sodium Sulfathiazole on Nasal Mucous Membranes: Results of Animal Studies, J. A. M. A. **119**:7-8 (May 2) 1942.

16 Fletcher, R.: The Caustic Action of Sodium Sulfathiazole, J. A. M. A. **117**:1204 (Oct. 4) 1941; Sodium Sulfathiazole: Its Caustic Action, California & West. Med. **55**:94 (Aug.) 1941.

twice a week. Every infant improved (table 2). The consensus of the attending physicians and nurses was that 80 per cent of the infants responded more quickly to treatment with the preparation than could be expected with substances previously used. With all but 2 babies the best results occurred after the first treatment and

TABLE 1.—*Data on Representative Patients Treated with Suspension of Sulfathiazole in Aqueous Solution of Paredrine Hydrobromide*

Patient	Age, Yr.	Complaint	Duration of Symptoms	Diagnosis	Paredrine-Sulfathiazole Preparation *	Period of Treatment	Response
M. M.	45	Cold	36 hr.	Acute rhinitis	5 drops q. i. d.	48 hr.	Cure
B. L.	40	Acute cold	48 hr.	Acute nasopharyngitis	5 drops b. i. d.	2 days	Cure
G. P.	1	Earache and discharge	10 days	Acute nasopharyngitis; acute otitis media with involvement of mastoid	3 drops b. i. d.	7 days	Disappearance of all symptoms
J. L.	6	Stuffy nose; conjunctivitis	3 mo.	Bilateral ethmoiditis	3 drops t. i. d.	3 wk.	Conjunctivitis cleared; ethmoiditis relieved
C. W.	27	Headache; stoppage of breathing	1 yr.	Acute sinusitis	Proetz method; 3 drops q. i. d.	2 wk.	Headache and sinusitis relieved
G. A.	50	Headache	2 yr.	Sinusitis	Proetz method; 3 drops b. i. d.	3 wk.	Headache relieved
D. G.	37	Headache; nasal discharge	Several years	Sinusitis	Proetz method; 3 drops b. i. d.	3 days 1 mo.	Headache relieved Improvement continued

* In this column, b. i. d. means twice a day; t. i. d., three times a day, and q. i. d., four times a day.

TABLE 2.—*Data on Twenty-Three Patients Treated with Paredrine-Sulfathiazole Preparation by the Proetz Method* *

Patient	Age, Mo.	Type of Discharge	Number of Treatments	Period of Treatment, Days	Response
1	4	Purulent	1	1	Good
2	5½	Purulent	3	8	Good
3	6	Watery	1	1	Excellent
4	7	Purulent	3	8	Excellent
5	7	Purulent	1	1	Good
6	10	Purulent	1	1	Excellent
7	11	Purulent	1	1	Excellent
8	11	Purulent	1	1	Excellent
9	12	Purulent	1	1	Excellent
10	13	Purulent	2	6	Good
11	13	Mucopurulent	1	1	Good
12	14	Purulent	1	1	Excellent
13	14	Thick; yellow	4	10	Good
14	15	Purulent	2	7	Fair
15	16	Thick; green	3	8	Excellent
16	16	Purulent	2	7	Good
17	16	Purulent	1	1	Good
18	17	Thick; green	3	8	Good
19	17	Thick; green	2	5	Good
20	17	Thick; green	2	5	Good
21	19	Thick; green	2	2	Good
22	21	Thick; green	3	8	Good
23	30	Purulent	2	5	Good

* Reported by Lieut. Richard P. Virnig (Medical Corps, United States Navy), St. Vincent's Hospital, Philadelphia.

within the first day. With all but 1 child the treatments were continued until the condition was satisfactory. This patient had only two treatments during the time allotted to this study. A satisfactory response was obtained within a week except in 1 case, in which four treatments were given in a period of ten days before a good reaction was shown. Use of the paredrine-sulfathiazole combination as many as seven times within three weeks in the Proetz treatment of a 14 month old child and twice a week with babies as young as 5½ and 7 months produced no untoward effects.

Two to 5 drops of the suspension of sulfathiazole in aqueous solution of paredrine hydrobromide has been instilled in adults two to four times daily for over eighty days without causing any undesirable effects. Burning or stinging was never observed, nor was there other evidence of ciliary damage. The patients' reactions have been enthusiastic. Many described the suspension as having soothing qualities.

Since any medicament used in the nose is apt to be washed or blown away, the application should be repeated at frequent intervals. It is desirable that the ambulatory patient be able to do this for himself at his convenience. Because of the relative insolubility of sulfathiazole and the small amount applied each time, there are no cumulative or systemic effects, and the suspension can be instilled as often as the patient wishes.

The paredrine-sulfathiazole preparation instilled into the nose during a cold acts both by constricting the mucosa and by rendering the medium unfavorable for supporting the multiplication of the bacteria which are susceptible to it. It also appears to enable the normal defense mechanism of the body to combat invading organisms successfully. It is safe to say that the use of this combination has avoided many of the dangerous sequelae to the common cold, such as pneumonia, otitis media and mastoiditis.

SUMMARY

A suspension of sulfathiazole in an aqueous solution of paredrine hydrobromide has been used in over 75 cases of rhinitis and sinusitis in the course of eight months. It has been successful in reducing nasal congestion and in decreasing or eliminating discharge, without evident injury to the cilia. There have been no untoward effects of any kind. Its use has shortened the course of infection for many patients and averted sequelae to colds. The amount employed is so small that it can be used repeatedly without systemic reactions from either the sulfathiazole or the paredrine hydrobromide component.

Personal communications from Dr. S. A. Goldberg, Rush Hospital, Philadelphia, and from Capt. Anthony Diodati, the Station Hospital, Aberdeen, Md., have substantiated my conclusions that a suspension of sulfathiazole in an aqueous solution of paredrine hydrobromide is effective as a bacteriostat and vasoconstrictor in the upper respiratory tract and has no unfavorable side effects.

323 South Eighteenth Street.

Case Reports

CALCIFICATION OF THE AURICULAR CARTILAGE

HORACE G. DWIGGINS, M.D., TUSKEGEE, ALA.

A review of the literature reveals that ossification or calcification of the auricular cartilage has been reported infrequently.[1] Sherrer,[2] after thorough study of his own case and an extensive review of the literature, was able to find only 40 cases reported from 1866 to 1931. His own case added another to the total number recorded by September 1932. In the same year Childrey[3] reported 8 cases and in 1938,[4] 7 additional ones. His article of 1938[4] cited 61 cases reported over a period of seventy-two years, apparently by 21 observers.

Aside from Fränklel[2] and Childrey, each of whom reported 15 cases, only Müller,[2] Lubber[2] and Osler[2] saw more than 1 case. It is also believed that all the patients were white persons with the exception of a Chinese reported on by Childrey.[4] The ages of most of the patients ranged from the fifth to the eighth decade.

The exact cause of the condition is not known, but as the literature accumulates, it seems that a history of frostbite is most frequently encountered. Childrey[4] concluded that this was the most frequent etiologic factor. Other causes[2] which have been mentioned are endocrine disturbances; senile keratosis; syphilis; perichondritis; inherent property of ossification of cartilage, and general debilitating diseases involving the blood supply to peripheral organs.

REPORT OF A CASE

History.—A well developed, well nourished, dark brown Negro aged 46 was admitted on April 24, 1942, to the facility at Tuskegee, Ala. He had had previous admissions to the Veterans Administration Facility, Columbia, S. C., beginning in 1938. The patient was illiterate and a common laborer; he had worked during prewar days in "saw mills."

The patient was married and the father of 5 children, 1 of whom died at 18 months of age, of "fever." His father was dead, the age and cause of death

---

From the Veterans Administration Facility.

Published by permission of the Medical Director, Veterans Administration Facility, who assumes no responsibility for the opinion expressed or the conclusions drawn by the author.

1. Ballinger, W. L., and Ballinger, H. C.: Diseases of the Nose, Throat and Ear, Philadelphia, Lea & Febiger, 1938, p. 627.

2. Sherrer, F. W.: Calcification and Ossification of the External Ears, Ann. Otol., Rhin. & Laryng. **41**:867-885, 1932.

3. Childrey, J. H.: Calcigerous Metaplasia in the Auricle, Arch. Otolaryng. **15**:883-884 (June) 1932.

4. Childrey, J. H.: Ossification in the Auricle: Seven Cases, Laryngoscope **48**:339-345, 1938.

being unknown. The mother was living and well. The patient had 2 brothers and 1 sister who were living and well; 2 brothers and 2 sisters were dead, the ages and causes of death being unknown. He did not know of a condition similar to his present complaint in any of his relatives.

The patient stated that he had had some of the illnesses of childhood but that as a young man he was strong and healthy and "could stand anything." He was first hospitalized in 1938 for an ocular condition and "running" of the right ear. His habits were moderate, and he denied having had venereal disease.

At the age of 17, while he was running machinery in a lumber mill, his ears were frostbitten daily during the cold part of the winter, but because he "could stand anything" he made no effort to protect himself. He could remember only a burning sensation about the ears after they "thawed out at night" and denied having a history of trauma or anything that could be considered an acute inflammatory process about the ears. Some time later he noticed his ears' feeling "stiff

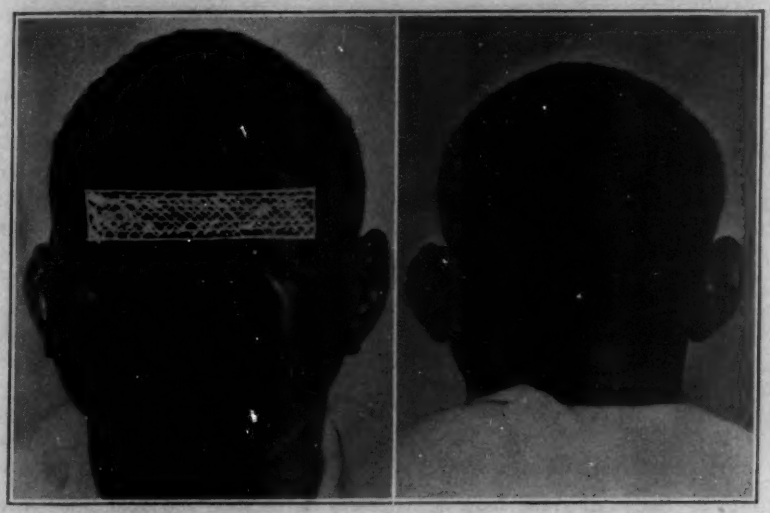


Fig. 1.—Prominence of the ears and wrinkling along the superior border of the left ear.

and hard and not bending like they used to." They had never hurt him; so he paid no attention to them. The process, he stated, was slow and took about four years to reach the state noted at the time of examination.

General Examination.—His general physical condition was good, and no physical defects were observed on examination except for residual iritis, bony hardness of both external ears, a mucopurulent discharge from the partly closed right external auditory canal and bilateral deafness.

Examination of the Ears.—The ears were prominently situated on the head. They appeared normal except for mild wrinkling along the upper margins. The overlying skin was warm and showed no evidence of a degenerative process, either recent or remote. Both ears felt bony and were not pliable in any portion, nor were they freely movable on the head. The right external canal was practically occluded, and a small amount of mucopurulent material was discharging

from it. The tympanic membrane could not be seen. The discharge was considered to be due to the damming up and secondary infection of the natural secretions. The left auditory canal was clean, and the drum gave off a normal light reflex. There was complete loss of hearing to air and bone conduction in both ears. The exact cause of the deafness was not known, nor had it been determined during previous hospitalizations. Although illiterate, the patient was rather apt and read the examiner's lips readily.

Roentgenologic Report.—Auricles: Extensive calcific deposits involved the superior portion of the helix on each side, to a considerably greater extent on the right than on the left, and extended on the right inferiorly to the level of the antitragus. Extensive deposits were seen in the antihelicus superior and inferior on both sides. Small deposits were seen in the helix on the left. The cartilage of the lobules remained unchanged. No calcific deposits were noted in

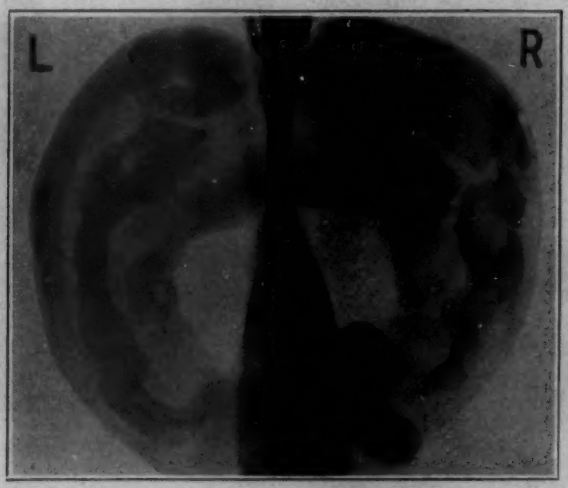


Fig. 2.—Roentgenogram, showing extensive calcification and ossification of the auricular cartilages.

the concha. Calcific deposits were observed in the crus antihelicus bilaterally. These deposits showed variation in density, with slight linear shadows which suggested trabeculations.

Impression: The roentgenologic diagnosis was bilateral calcific deposits within the cartilage of the ear.

Laboratory Reports.—The Wassermann, Kahn and Eagle reactions were negative. Urinalysis showed no deviation from the normal limits. A hemogram revealed 4,760,000 red cells, 4,900 white cells and 80 per cent hemoglobin. The sputum was negative for tubercle bacilli. The discharge from the ear showed pus cells and debris.

This case seems to conform in every way to the cases reported in the literature. Definite hardening of the auricular cartilage follows a history of repeated frostbite. In this case the condition was recognized quite by accident during a routine examination and excited interest because of the

apparent extensiveness of the process. According to Sherrer,[2] Childrey [3] and Higbee,[5] this is the most usual method of recognition.

The etiologic factor in this case, namely repeated frostbite, seems well established; the possibility of the other causes cited in the literature is ruled out, there being no history of trauma, syphilis, chronic inflammation, debilitating disease or endocrine disturbance. The patient was deaf in both ears and had a chronic infection of the right ear but both conditions seem to have developed some time after the hardening process had reached its maximum intensity. Sherrer,[2] Childrey [4] and Higbie [5] mentioned the presence of abnormal contents in the external auditory canals in their cases and attributed the discharge to destruction of the glands lining the canal, possibly because of poor blood supply. Higbie [5] and Childrey [2] spoke of loss of auditory acuity and deafness in their cases and attributed it to possible otosclerosis. However, their deductions were not conclusive in this respect. It would seem, although the cause is not fully understood, that patients with ossification of the auricular cartilages usually suffer some degree of deafness in time.

All authors except Fränklel and Childrey stated the opinion that calcification and ossification of the auricular cartilage is rare. In Childrey's cases the roentgenographic evidence was conclusive, but Fränklel [4] reported cases of calcified deposits which, in many instances, were no larger than a mustard seed. It appears that even though the condition has been infrequently described, it may not be so very rare, since 2 observers have found and reported 15 cases each. On the other hand, it is infrequent enough, since most observers have seen only 1 case in a lifetime.

A case of this type brings to mind that men participating in the present war, being often exposed to very low temperatures, may be expected to manifest ossification and hardening of the auricular cartilages in a small number of instances. Also, since there is a pathologic process which interferes with auditory acuity, deafness may be expected to result after an indefinite period.

CONCLUSIONS

A case of calcification and ossification of the auricular cartilages in a Negro is reported. This case adds another to the total number of instances of this rare condition.

The condition most frequently follows frostbite of the ears.

Deafness develops in most cases of this condition.

Veterans Administration Facility.

5. Higbee, D. R.: Calcigerous Metaplasia of the Auricular Cartilage, Arch. Otolaryng. **14**:70-79 (July) 1931.

Clinical Notes; New Instruments and Technics

A NEW METHOD FOR REMOVING WAX FROM THE EAR

FRANCIS L. WEILLE, M.D., BOSTON

A fairly efficient method of removing wax from the ear is the use of a metal suction tip with strong negative pressure; up to 25 pounds (11.3 Kg.) of suction may be required. A no. 1 silver eustachian catheter can easily be converted into such a tip, although a smaller tube is preferable. The figure shows the type I use. This was made by a surgical instrument company in Boston, at a cost of $1. An ordinary suction machine with a gage to regulate the negative pressure up to 30 pounds (13.6 Kg.) is desirable, but not essential.

Possible injury to the drum is so easily avoided by gentleness and mechanical skill at the fundus of the canal as to be of academic interest only.

Suction tip for removing wax from the ear.

Cerumen in the external auditory canal offers such a simple problem in therapeutics as to arouse only mild interest among ear specialists. However, general practitioners find "washing out the ear" to be a common task, and occasionally, because of a known open perforation in the drum or because of eczema of the canal, the patient must be sent to a specialist for mechanical removal of the wax by a "dry" method. It is well known that the penalty for "wet" removal of wax, by syringing with water, in certain cases of eczema may be a "weeping" membrane of the external auditory canal for weeks or months, or, if there is a dry middle ear with an open perforation in the drum, syringing of the canal with water is likely to produce intractable drainage from the middle ear. For such conditions the "suction method" is valuable.

The technic is efficient with soft, moderately viscous or even greatly inspissated cerumen. With hard, dry wax, loosened if necessary by a cerumen hook, incredibly large masses of cerumen can be withdrawn, attached to the tip of the suction tube. Here, suction offers the advantage of withdrawal of material from the canal, as contrasted with a possible pushing action toward the drum of any type of grasping forceps, especially in unskilled hands. Furthermore, any wax not

hard enough to be easily engaged with a cerumen hook can be drawn through a suction tip. It is the purpose of this report to emphasize this fact.

The "suction method" which has been described requires no special apparatus other than that found in a good general hospital or in the office of the average ear specialist.

395 Commonwealth Avenue.

TREATMENT OF CHRONIC SINUSITIS WITH IODIZED OIL

J. Jerome Littell, M.D., Indianapolis

The injection of iodized oil into the sinuses has been carried out for many years—being first, and more popularly, used as a diagnostic means. Recently Goodyear [1] has laid emphasis on the value of the oil as a therapeutic agent for disease of the sinuses. His pronouncements have given rise to a certain amount of controversy. It is with this in mind that I presume to relate my own observations.

Like Goodyear, many who have used the iodized oil have noticed that patients volunteered the information that the side on which the oil had been injected for diagnostic purposes felt vastly relieved after the "treatment." This was surprising, as no attempt at formal treatment had been made. This effect was often obtained when the condition was bilateral and similar shrinkage had been applied to each middle meatus. The feeling was that the oil was a factor in the improvement, rather than the shrinkage of the ostium, which Van Alyea had suggested might be responsible. However, I did not routinely use the agent generally employed (which was 40 per cent iodized poppyseed oil in 50 per cent combination with liquid petrolatum), as occasionally I noted an annoying acute exacerbation of the symptoms on the side of injection, suggesting that the oil was irritating to some people. For the past three years, however, I have been using 27 per cent iodized and 7.5 per cent chlorinated peanut oil [2] in 50 per cent combination with oil of sesame.

This combination of oils is more readily injected because of its lower viscosity. It is of sufficient density to cast a sharp shadow roentgenographically, is less expensive than the undiluted oil and appears to have therapeutic value. There have been no unpleasant vasomotor symptoms in the nose after its use, except in 2 patients who were found to be sensitive to iodine in any form. In 1 of these patients there developed a watery edema involving the subcutaneous tissues beneath the mandible from one side to the other for nearly 1 inch (2.5 cm.). The patient afterward announced that a similar reaction had occurred when he had taken iodine by mouth. This reaction seems to point clearly to the absorption of iodine by the tissues, even though, as Proetz remarked, the solution employed is chemically inert, in fixed combination, and the iodine is not given off separately.

From the Department of Otorhinolaryngology, Indiana University School of Medicine.

1. Goodyear, H. M.: Use of Iodized Oil in Treatment of Infections of the Nasal Antrum, Arch. Otolaryng. **34**:1133 (Dec.) 1941.

2. The preparation used was iodochlorol (G. D. Searle & Co., Chicago).

In my experience the most spectacular results have been noted in cases in which a partial allergic or vasomotor background may be suspected. The mucous membrane usually is not the angry red which one associates with true infection, but is more pallid—being overmoist and shining, a membrane which does not

Fig. 1.—My apparatus for washing out sinuses. It has simplified the technic, so that no assistant is needed.

A, enameled pan, over which the patient leans. *B*, a 1 gallon (4 liter) picnic jug. When half filled with solution it will stay sufficiently warm all day. *C*, pressure gage. This is not absolutely necessary. I never use a pressure of over 5 pounds (2.3 Kg.). *D*, movable base (on casters). *E*, three way valve. When the valve is moved one way, solution flows up from the jug and out the rubber tube. When it is turned another way, air, from the compressed air space above the fluid, comes out the rubber tube and blows the solution out of the sinus. *F*, ball valve. At its end is the female coupling, which connects with the Deviliss cut-off. Compressed air put in here remains in the upper part of the jug. *G*, metal tubes, which pass through the cap down into the jug. One is short and goes only into the air space below the cap; the other goes to the bottom of the jug. *H*, slip joint fastened onto the end of the rubber tube, with a shield at its proximal end. Distally it fits into the Littell cannula. A lock joint is not used, so that excessive pressure cannot be built up in the sinus. In the only case in my experience in which air embolism was feared, I used all the pressure I could muster to force the flow in a refractory antrum. *J*, turn table, which allows the apparatus to be turned on the pedestal.

shrink well—the picture suggesting an oversupply of serum in the submucosa. I have attempted to substantiate this observation by the determination of eosinophils in nasal smears and have found the results inconclusive. It is an impression which one must often get from clinical observation only.

My experience has also been most favorable in cases of the subacute or chronic condition. The acute disturbances do quite as well with lavage, or lavage followed by the injection of a 5 per cent solution of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole). But lavage is less dramatic with low grade disturbances, and it is here the injection of oil occasionally seems to have a place.

It is my practice to lavage the sinus through a cannula placed in the middle meatus—either through the normal ostium and the accessory ostium or through the membranous portion at the tip of the uncinate process. When pus is present in any amount it seems best first to lavage the sinus and then to blow out the irrigating solution (physiologic solution of sodium chloride) in order to obtain a comparatively dry sinus before injecting the oil through the same cannula.

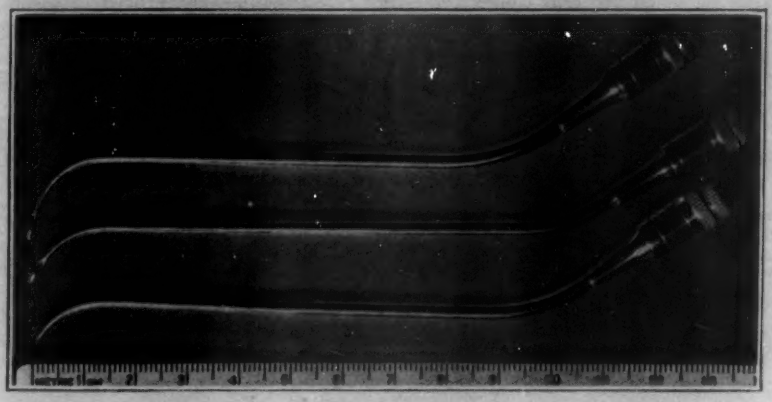


Fig. 2.—The cannulas are arranged in the order of their importance from below upward. The lower one will enter the greater majority of antrums; the upper is useful in the unusual case, in which the lateral wall of the middle meatus has to be reached around a heavily projecting inferior turbinate body.

The aperture at the large end is a slip joint, so that excessive pressure will not be built up during the lavage. I never exceed a pressure of 5 pounds and use even that with caution.

The cannula (made by Storz) which suits my purpose best (where is the specialist who will not insist on designing his own instruments?) is a modification of ones previously developed. The instruments, of shapes which allow entrance into almost any antrum, are pictured here. The cannula is rigid; the terminal, or delicate, portion is of stainless steel, is small enough to pass through constricted areas without undue force, does not obstruct vision and is strong enough to break through the wall if necessary. The tip is semisharp to make the last procedure more easy. The lumen is of sufficient size to allow liquids to pass with ease.

In casting about for a satisfactory explanation of the improvement in cases of chronic sinusitis, I have felt that often the good effect is due to mild stimulation

of the mucous membrane not only of the maxillary sinus but of the superimposed ethmoid cells. The diagnosis may be chronic ethmoiditis only, with no clinical or roentgenologic evidence of antral disease; yet the patient will report a copious discharge, usually postnasal, of mucopurulent secretion for some time after the injection, with much subjective relief. A second possibility is that the iodine, which in vitro seems to be in close chemical combination, is really available to the tissues and helps to liquefy the mucus and stimulate its production (use of iodides for bronchitis and asthma) or has some beneficial effect on disturbances of the thyroid, which are often present with waterlogged nasal membranes.

This treatment is no panacea. A great many patients will report no unusual improvement after its use. On the other hand, there are persons who, having been subjected to other methods of treatment, will insist on further injection of the oil as being much the most helpful therapy that has been employed.

CONCLUSIONS

The use in the large sinuses of 27 per cent iodized and 7.5 per cent chlorinated peanut oil in 50 per cent combination with oil of sesame has proved of value in the treatment of low grade rhinosinusitis.

Many of the cases in which this therapy is of value seem to fall into the group in which chronic infection may be encouraged by defects in the local vasomotor apparatus (subclinical allergic states; glandular dyscrasia).

While this treatment does not replace surgical correction, it is often helpful in bringing about a condition of comparative comfort.

23 East Ohio Street.

Progress in Otolaryngology

Summaries of the Bibliographic Material Available in the Field of Otolaryngology

---

SUPPURATIVE OTITIS MEDIA AND ITS COMPLICATIONS

ROBERT C. MARTIN, M.D.

DOROTHY DANNO, M.D.

AND

J. F. SKELLY, M.D.

SAN FRANCISCO

The literature on otitis media and its complications, with the omission of meningitis, is reviewed from January 1941 to July 1942. As might be expected, the paramount interest lies in chemotherapy.

GENERAL CONSIDERATIONS

Day [1] presents an interesting paper on cholesteatoma, in which he states that cholesteatoma resulting from ingrowth of epithelium through the drum membrane should be called pseudocholesteatoma. The lesion may be primary, arising from a defect of Shrapnell's membrane, regarding which there is a difference of opinion, or secondary (more common), from the ingrowth of skin from the external canal into the middle ear, replacing destroyed mucosa. He believes that retraction of Shrapnell's membrane is caused by a closing off of the attic from the middle ear due to the entrance of irritating fluids or foreign material into the latter. One of the causes is the prone-back position in which bottle-feeding infants are placed.

Diamant [2] studied the air cell systems in 905 patients, of whom 320 were normal and 585 had had otitis media (chronic in 50 per cent). He noted moderate regression in size after the age of 30. Patients with chronic otitis showed mastoids one-fourth as large as the average. He states that acute otitis develops in ears in which the air cells average about four-fifths the normal size, while it rarely occurs in persons with the largest cell systems. (The last observation is at variance with our experience.) Examination of normal persons showed that the mastoids usually have a nearly equal development on the two sides. Persons with chronic otitis had a small mastoid on the uninvolved, as well as on the involved, side.

CHRONIC OTITIS MEDIA

Lillie [3] recommends hospitalization of the patient with acute infection of the upper respiratory tract. He advises adequate myringotomy, with the patient under general anesthesia, and culture of material from the ear. Irrigation is used only when the discharge is thick, to facilitate drainage. Differentiation of the type of

---

1. Day, K. M.: Primary Pseudo-Cholesteatoma of the Ear, Arch. Otolaryng. **34**:114 (Dec.) 1941.

2. Diamant, M.: Otitis and Air Cell Systems, Arch. Otolaryng. **34**:24 (July) 1941.

3. Lillie, H. I.: Treatment of Acute and Chronic Suppurative Otitis Media, J. A. M. A. **115**:506 (Aug. 17) 1940.

mastoiditis present (osteothrombotic or suppurative) is made on the kind of discharge, the roentgenogram and the blood picture. Lillie feels that the general appearance of the patient is a good index to his condition (an excellent point, which is not sufficiently emphasized in present day teaching).

Four types of acute otitis media may be differentiated:

1. Anteroinferior perforations overlying the eustachian tube and no symptoms other than discharge.

2. Central perforation and pathologic alteration in the membrane over the promontory, with little pathologic change in the attic. This form responds well to local treatment.

3. Perforations of the attic, with evidence of necrosis of bone and cholesteatoma. In addition to the foul-smelling discharge, such symptoms as headache and vertigo may be present.

4. The same pathologic picture as that in type 3 except that symptoms indicating impending intracranial complications are present.

Morrison [4] presents a good outline of the modern conception of the pathology, diagnosis and treatment of chronic suppurative otitis media. He again emphasizes the dangers of cholesteatoma and expresses the belief that operation is always indicated for such a condition. To preserve hearing he employs the modified radical mastoidectomy, but if the hearing is poor or intracranial complications threaten or are present he prefers the full radical mastoidectomy. He shortens the period of healing by using multiple pinch skin grafts on the tenth to the fourteenth day, inserting them through the external meatus. Primary skin grafts rarely take.

The value of the endaural approach in cases of chronic suppurative otitis is emphasized by Shambaugh,[5] in a report of 100 consecutive cases of chronic suppurative otitis media encountered in office practice. He notes that in cases of primary acquired cholesteatoma in which surgical treatment was required the postoperative hearing was never worse and that in 67 per cent it was improved when the Bondy modification of the radical mastoidectomy, with the endaural approach, was done.

A case of generalized tetanus complicating a chronic purulent infection of the middle ear is reported by Rosen.[6] The bacilli were recovered from the middle ear.

The incidence of tuberculous otitis media after thoracoplasty led Adams [7] to study the patency of the eustachian tube by means of a mercury manometer to which was attached a nasal tube held firmly in one side of the patient's nose, while the other side of the nose was kept closed. An ordinary diagnostic tube led from the patient's to the examiner's ear. The click heard when the air entered the middle ear indicated the pressure on opening of the eustachian tube. The minimal normal pressure, as noted by Perlman, was 20 mm. of mercury. When less pressure was required Adams used boric acid, 1 part, and salicylic acid, 4 parts, in the eustachian tube to diminish its patency. He gives a good description of the objective observations. In the first stage there is congestion

4. Morrison, W.: A Modern Conception of the Pathology, Diagnosis and Treatment of Chronic Suppurative Otitis Media, Ann. Otol., Rhin. & Laryng. **51**:5 (May) 1942.

5. Shambaugh, G. E.: Treatment of Chronic Suppurative Otitis Media with the Use of the Endaural Approach for the Radical Mastoidectomy, New Orleans M. & S. J. **94**:105 (Sept.) 1941.

6. Rosen, S.: Otogenous Tetanus, Laryngoscope **51**:892 (Sept.) 1941.

7. Adams, J. G.: Tuberculous Otitis Media: A Complication of Thoracoplasty, Ann. Otol., Rhin. & Laryng. **51**:209 (March) 1942.

of the membrana tympani, more severe in the pars flaccida, which cannot be differentiated from acute catarrhal otitis media. This lasts about one week, when the second stage occurs, characterized by a bulging drum, grayish white, with individual blood vessels crossing the bulging portion from the periphery toward the umbo and along the process of the malleus. The short process is prominent, and the contour is that of an inflated ear. This stage may last from two weeks to several months, when the third stage, with watery discharge and epithelial desquamation, occurs. Smears are more often positive for tubercle bacilli early in the third stage, before secondary infection is prominent. Roentgenograms show pneumatization and periantral sclerosis, while with other types of chronic purulent otitis media nonpneumatization and sclerosis are noted.

In a comment on the operative treatment of mastoiditis, Blashki and Clowes [8] state that in cases of chronic otitis rarely, if ever, is there indication for the classic radical operation, even in the presence of cholesteatoma. (This statement does not agree with our experience.)

ACUTE OTITIS MEDIA

Barkhorn,[9] in a study of the acutely involved mastoid before and after operation, concludes that failure to heal after mastoidectomy is due to (1) excessive widening of the tympanic antrum, (2) excessive smoothing of the operative cavity, (3) improper management of the zygomatic root, (4) continuing necrosis of the inner table of the bone and (5) suppuration beyond the confines of the mastoid process. He emphasizes the value of preoperative and postoperative roentgenographic studies in showing (1) the type of mastoid, (2) periantral density, which he believes is increased in patients with perisinal abscess, (3) the visibility and sharpness of outline of the lateral sinus wall, (4) the condition of the trabeculae and cells and (5) the appearance of the apex of the petrosa. We feel that roentgenographic studies are in order to establish the appearance and topographic relations of the mastoid, knowledge of which will make for completeness of operation and allow one to work with greater assurance that no cell group will be overlooked. Should complications follow, the earlier roentgenograms give a basis with which changes may be compared. We have failed to note that periantral density is suggestive of perisinal abscess.

The relation of infantile mortality and infantile mastoiditis was studied by Leathart.[10] In a series of 72 autopsies in the Royal Liverpool Children's Hospital during "the last two years" it was shown that mastoiditis was undiagnosed in 59.6 per cent of cases. Sixty children died of air-borne and 12 of food-borne infection. The rate of recovery was 80 per cent in 150 cases in which operation was performed. Like Day, Leathart feels that an infant who is ill with air-borne or food-borne infection should always be propped up when fed and should not be allowed to lie on its back, because of the danger of aspiration due to the short, wide eustachian tube.

Bacteriologic studies were done in cases of otitis media and mastoiditis by Neter, Neuberger and Short.[11] The hemolytic streptococcus occurred in 80 per

8. Blashki, E. P., and Clowes, A. L.: Operative Treatment of Mastoiditis, M. J. Australia **1**:443 (April 12) 1941.

9. Barkhorn, C. W.: The Acutely Involved Mastoid (Without Complications) Before and After Operation, Arch. Otolaryng. **34**:69 (July) 1941.

10. Leathart, P. W.: The Relation of Infantile Mastoiditis to Infantile Mortality, J. Laryng. & Otol. **56**:320 (Sept.) 1941.

11. Neter, E.; Neuberger, E., and Short, L. M.: Otitis Media and Mastoiditis in Children, Arch. Otolaryng. **35**:631 (April) 1942.

cent of the cases of mastoiditis and in 30.5 per cent of the cases of otitis media. Pneumococci occurred in 13 per cent of the cases of mastoiditis and in 22 per cent of the cases of otitis. Staphylococci, the pathogenicity of which was determined by the plasma coagulose test, occurred in 1 per cent of cases of mastoiditis and in 36 per cent of cases of otitis media. Mixed infection occurred more often in cases of otitis media.

Stuart [12] reviews the cases of acute suppurative otitis media occurring during a five year period, from 1935 to 1939. The paper is a statistical study on the complications, age incidence and bacteriologic features and is well done. There were 754 cases of otitis media and 325 cases of mastoiditis. Eighteen deaths occurred, 16 of which were due to meningitis, 1 to pneumonia and 1 to cerebellar abscess. Emphasis, again, is placed on Pneumococcus type III as a dangerous organism. Although observed in only 71 cases of acute purulent otitis media, this organism accounted for one third of the deaths and for 30 per cent of the intracranial complications. Stuart noted that in 1939, when sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) was used, no complications other than mastoiditis occurred, while previously additional complications had been present in one third of cases. This period seems to be too short for one to draw conclusions as to the efficacy of the sulfonamide compounds. The virulence of organisms waxes and wanes.

The association of Pneumococcus type III with otitis media was studied by Persky,[13] who reviewed 307 cases in which mastoidectomy was performed from June 1, 1935 to Jan. 1, 1940 at the Graduate Hospital of the University of Pennsylvania and the Mount Sinai Hospital in Philadelphia. In 190 cases (154 cases of acute and 36 cases of chronic otitis) positive cultures were obtained. Pneumococcus was found in 33, or 17.3 per cent, of the positive cultures and in 10.8 per cent of the 307 cases of mastoidectomy. The type III pneumococcus occurred in 26 cases. Five deaths, all from meningitis, occurred in the 33 cases of pneumococcic meningitis. Persky states that the pneumococcus seems to have a predilection for the extensively pneumatized bone. In commenting on Diamant's statement that acute otitis rarely develops in mastoids showing the largest cell system, we noted that such was not our experience. Apparently, it was not Persky's, either. The sulfonamide compounds are held to be of little value for acute otitis and mastoiditis due to the pneumococcus when actual necrosis of the bone has occurred. (We believe this is generally accepted for all types of infection.)

Cooper [14] reports an interesting case of acute mastoiditis complicated by Schüller-Christian disease. The occurrence of acute streptococcic otitis and mastoiditis in a 3 year old girl was ascribed to impaired nutrition of the temporal bone, roentgenograms showing the presence of the punched-out areas (characteristic of this metabolic disturbance) in the body and great wings of the sphenoid bone, the orbital roof and the mastoid portions of the temporal bone. The frequently associated diabetes insipidus was present. After mastoidectomy roentgen therapy was instituted; the wound healed; the symptoms of diabetes insipidus disappeared, and follow-up roentgenograms showed recalcification of the involved areas of the skull.

---

12. Stuart, E. A.: Acute Suppurative Otitis Media: Review of Cases Encountered in a Five Year Period, Arch. Otolaryng. **33**:734 (May) 1941.

13. Persky, A. H.: Pneumococcus Type III in Otitic Infections, Arch. Otolaryng. **34**:473 (Sept.) 1941.

14. Cooper, K. G.: Acute Mastoiditis Complicated by Schüller-Christian Disease, Arch. Otolaryng. **33**:1028 (June) 1941.

Involvement of the "jaw joint" with acute suppurative otitis media is reported in 3 cases, all those of children, by Shambaugh.[15] The pus lay between the bone and the joint capsule. The routes of infection are as follows: (1) by metastasis, as in thrombosis of the lateral sinus; (2) by bony erosion of the infected zygoma, and (3) by thrombophlebitis of the anterior tympanic veins, which run from the middle ear through the petrotympanic fissure to join the plexus around the jaw joint. Lillie and Simonton[16] report 2 cases. The route of extension is given as the glaserian fissure. The differential diagnosis between this condition and so-called zygomatic abscess lies in the more severe trismus and pain with abscess of the mandibular joint; the degrees of swelling are the same. Roentgen examination usually gives evidence of the presence of mastoid cells beyond the anterior root of the zygoma in cases of abscess of the zygomatic fossa, but not necessarily in cases of abscess of the mandibular fossa. Shambaugh drains the abscess by external incision, while Lillie and Simonton expose the lesion as follows:

> The incision employed for a mastoid operation is extended above and around the upper attachment of the ear. Cutting into the temporal muscle is avoided, after this muscle has been exposed, by making an incision to the bone along the lower aspect of the zygoma to a point just beyond the articular eminence of the mandibular fossa. The temporal muscle and fascia are freed upward sufficiently, and the structures below are separated downward to expose the anterior wall of the bony canal and the curve of the middle root of the zygoma, which forms the fossa on the inner aspect. With a sharp chisel or gouge, a wedge of bone is removed in the direction of the middle ear to the site of the opening of the eustachian tube into the middle ear. The articular capsule is then separated from the mandibular fossa with a suitable periosteal elevator. Drains are inserted, and the upper part of the wound is dressed and kept open until healing takes place. If the abscess has extended into the pterygomaxillary fossa, the question of through and through drainage must be settled at the time. If secondary drainage is deemed necessary, an incision involving the parotid gland must be avoided because of the danger of persistent parotid fistula.

CHEMOTHERAPY

The literature on chemotherapy is large in proportion to the total number of articles on otitis.

Local use of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) crystals or another of the sulfonamide compounds in mastoid wounds followed by tight closure is advocated by Livingston.[17] He claims a shortened postoperative period and primary healing in nearly all cases. Our own experience was that sulfathiazole formed clumps that acted as foreign bodies and that their removal was necessary to secure healing. Use of sulfanilamide gave a clean wound (we did not use primary suture) but did not shorten convalescence. Recent investigations show that sterilized crystals only should be used on account of the danger of tetanus.

In a case reported by Bryant[18] dirty granulations which were increasing after operation were cleaned with the local use of a sulfonamide compound. He also notes cases of chronic otitis and mastoiditis in which the condition subsided after local chemotherapy. Our experience bears out this observation.

Guerry and Putney[19] report 40 cases of surgical mastoiditis, in half of which the operative wound was packed with gauze soaked with a sulfathiazole paste, in

15. Shambaugh, G. E.: Involvement of the Jaw Joint in Acute Suppurative Otitis Media, Arch. Otolaryng. **33**:975 (June) 1941.

16. Lillie, H. I., and Simonton, K. M.: Abscess of the Mandibular Fossa Secondary to Otitis Media, Arch. Otolaryng. **34**:485 (Sept.) 1941.

17. Livingston, G. S.: Local Sulfonamide Therapy in Acute Mastoiditis, J. A. M. A. **117**: 1081 (Sept. 27) 1941.

18. Bryant, F. L.: Local Use of Sulfanilamide in Otogenic Infections, Arch. Otolaryng. **35**:281 (Feb.) 1942.

19. Guerry, DuP. III, and Putney, F. J.: Local Use of Sulfathiazole in Management of Simple Mastoidectomy Wounds, Arch. Otolaryng. **35**:399 (March) 1942.

addition to the routine treatment. No drug was given orally, and no attention was paid to the nature of the causative organism. The drug-treated wounds healed more rapidly; the discharge from the middle ear ceased almost at once, and drainage of the wound persisted nine to thirty-six days (average fourteen and nine-tenths days). In the untreated controls the discharge from the middle ear persisted for some time, and the mastoid drained from seventeen to sixty-six days, an average of thirty-one and six-tenths days. (There would seem to be less danger of the drug's clumping in the wound when this method is used.)

In a series studied at Children's Hospital in Philadelphia by Frauenberger,[20] the incidence of otitis and mastoiditis since the advent of chemotherapy showed no decrease from that for the past ten years. He makes the interesting observation that in one third of the cases in which there was spontaneous rupture mastoidectomy was later required, while in one ninth of the cases in which paracentesis was done mastoidectomy was necessary. In contrast, Jessiman[21] reports 7 cases of acute suppurative otitis media with bulging of the drum membrane occurring in children which was successfully treated (without paracentesis) by the early use of a sulfonamide compound.

Bilchick and O'Kane[22] recommended the routine administration of a sulfonamide compound in cases of acute otitis media, but stress the importance of one's not neglecting indications for myringotomy and mastoidectomy as they arise, since excessive reliance on the drug will result in frequent disasters. They state that 55 children with 74 inflamed ears (unselected cases) were treated in the outpatient department. Control series were treated by routine conservative measures. The number of mastoidectomies was not reduced by use of the drugs. (This is a welcome check on the present wave of enthusiasm for the routine use of the drugs.)

Ganz and associates[23] report an incidence of 2.94 per cent of cases of surgical mastoiditis among clinic patients who were not treated with sulfanilamide and 2.57 per cent among private patients receiving sulfanilamide. The drop in the incidence of surgical mastoiditis over a ten year period in the group of private patients began in 1938 and is due chiefly, they believe, to the use of sulfonamide compounds. No decrease in the incidence was noted in the public ward, where these drugs were not used. In 39 of 40 patients the use of immune serum for patients not clinically convalescent in forty-eight hours after myringotomy and the administration of a sulfonamide compound prevented surgical mastoiditis. Nine patients in this group had hemolytic streptococci and 31 pneumococci.

Williams and associates[24] cite Kopetzky and Hadjopoulos thus:

> . . . . in acute mastoiditis caused by hemolytic streptococci variations occurred from year to year in the severity of the infection and also in the mortality rate. For a period of years these variations were characterized by regular periodicity; the authors suggested that a major cycle would consist of a period of five to six years.

Williams and his co-workers report that a slightly more favorable result with regard to the development of mastoiditis was obtained in patients with infections due to hemolytic streptococci who were treated with sulfanilamide. In the group

20. Frauenberger, G. S.: Infections of the Middle Ear as Related to Other Respiratory Infections, Arch. Otolaryng. **33**:1105 (June) 1941.

21. Jessiman, J. B.: Chemotherapy and Conservation in Acute Suppurative Otitis Media, Brit. M. J. **1**:399 (March 15) 1941.

22. Bilchick, E. B., and O'Kane, G. H.: Sulfonamide in Otitis Media in Children, Laryngoscope **51**:197 (Feb.) 1941.

23. Ganz, R. N.; Lyons, C., and Ferguson, C. F.: Chemotherapy and Serotherapy of Acute Otitis Media, Ann. Otol., Rhin. & Laryng. **50**:1185 (Dec.) 1941.

24. Williams, H. L., and others: Sulfonamide Therapy for Acute Otitis Media and Mastoiditis, Ann. Otol., Rhin. & Laryng. **50**:336 (June) 1941.

of patients with pneumococcic infections therapy with sulfonamide compounds produced an unquestionable advantage. The authors point out that in a patient receiving treatment with a sulfonamide drug little or no biologic resistance develops; thus, on discontinuance of the therapy, if surgical intervention becomes necessary, several days should be allowed to elapse, so that the body may produce some localization of the infection. Surgical intervention undertaken too soon may produce a severe systemic reaction, caused by dissemination of infection. In 10 per cent of the patients who received "adequate" doses there developed toxic reactions which necessitated discontinuance of the drug. The authors believe that treatment with the sulfonamide compounds is a hospital procedure.

Lindsay [25] believes that the widespread use of these drugs has reduced the incidence of surgical mastoiditis. He recognizes, however, that it has been demonstrated in several cases that large foci of necrosis or abscesses may resist chemotherapy.

In a paper on acute surgical mastoiditis with reference to postoperative hearing and the role of sulfanilamide and its derivatives in therapy, Priest [26] notes: (1) There is a reduction in deaths from complications; (2) mastoiditis can and does develop with sulfonamide drug therapy; (3) the reports on treatment with the drugs are variable; (4) toxic effects are possible, and masking of complications is sometimes noted. Postoperative hearing sometimes approaches normal. (Certainly, we have found hearing to be normal in patients tested five to ten years after operation.)

Cohen and Galpern [27] report a case of meningitis due to Staphylococcus albus haemolyticus with otitis media and mastoiditis in which sulfathiazole was administered intravenously after hematuria had developed with sulfapyridine therapy. Improvement followed, but relapse occurred after withdrawal of the drug. Finally, 100,000 international units of polyvalent staphylococcus antitoxin was given in divided doses along with the sulfathiazole. The patient recovered.

A series of 103 patients was reported on by Bilchick and O'Kane.[28] Fifty-five received a sulfonamide compound and 48 no drug. The period of aural discharge averaged seven days for the treated and twelve and six-tenths days for the untreated patients. Mastoiditis developed in 6 of the treated and in 4 of the untreated patients. An interesting point is that patients with recurrent mastoiditis were not benefited by chemotherapy. There were no complications or fatalities in either group. In a later communication,[29] the authors report on 80 patients, of whom half were treated with a sulfonamide drug and half served as controls. The untreated patients remained well, while 5 of the treated patients had an early and 3 a late recurrence of the discharge. The recurrences were said to be due to the flare-up of a latent process caused by failure of development by the body of immunity to the organism. The authors suggest adjuvant therapy with antiserum and transfusion from immune donors.

---

25. Lindsay, J. R.: Chemotherapy in the Treatment of Complications of Acute Middle Ear Suppuration (Petrositis and Meningitis), Ann. Otol., Rhin. & Laryng. **50**:159 (March) 1941.

26. Priest, R. E.: Acute Surgical Mastoiditis, with Reference to Postoperative Hearing and the Role of Sulfanilamide and Its Derivatives in Therapy, Arch. Otolaryng. **35**:250 (Feb.) 1942.

27. Cohen, B. M., and Galpern, M.: Sulfathiazole for Treatment of Staphylococcic Meningitis, Arch. Otolaryng. **33**:415 (March) 1941.

28. Bilchick, E. B., and O'Kane, G. H.: Sulfanilamide in Otitis Media in Children, Laryngoscope **51**:197 (Feb.) 1941.

29. Bilchick, E. B., and O'Kane, G. H.: Sulfanilamide in Otitis Media in Children, Laryngoscope **52**:341 (May) 1942.

Fowler Jr.,[30] in a report on the bacteriologic aspects of acute otitis media and the course of the disease under the new and the old treatment, states that the infection is self limiting and that, as such, about 80 per cent of unselected patients recover in less than four weeks. More patients would avoid mastoidectomy if their physicians would treat them expectantly for a longer period. Fowler feels that a large percentage of cures reported to have resulted from chemotherapy are due to spontaneous remission, rather than to the medication. Chemotherapy is indicated for patients with acute suppurative otitis media who are not doing well after ten days, and should be vigorously instituted. This form of therapy should be considered as a serious measure—in the same class as surgical treatment—and should always be given in the hospital, with adequate checks and never without careful bacteriologic study. If after two to three days the picture is unchanged, another method of treatment, usually surgical, is called for.

SINUS THROMBOSIS

Congenital absence of the cranial venous sinuses on the right side was reported by Williams and Hallberg.[31] The condition was observed in an 11 month old child with acute mastoiditis who presented an elevated, septic temperature. Culture of the blood yielded Streptococcus haemolyticus. Exploration revealed an epidural abscess but no evidence of the sigmoid or lateral sinuses or the superior or inferior petrosal sinuses. There was no persistent petrosquamosal suture. Recovery followed drainage of the abscess. The authors suggest that the septicemia may have been due to involvement of the lateral sinus of the opposite side, the venules of the dura or the diploic veins, though there was no evidence of encephalitis or osteomyelitis. The presence of a jugular bulb, however, was not eliminated.

Hubert[32] presents 119 cases of sinus thrombosis, 95 of which occurred before the advent of the sulfonamide drugs and 24 since. He concludes that the percentage of recoveries has more than doubled with use of the drugs.

An analysis of the changes in the ocular fundus in cases of otitic sinus thrombosis was made by Friesner,[33] who states that no satisfactory cause of the papilledema associated with this condition has been established. Venous stasis is not the cause, since (1) in many cases the papilledema occurred before operation; (2) in some cases in which swelling of the disks occurred postoperatively it came on four, five or six weeks after operation (ligation of the jugular vein), and (3) in cases of complete block of the venous sinuses it is obvious that ligation of the jugular vein would not produce stasis. Increased lumbar pressure is not the cause, as in no cases which Friesner reports was there an increase. A toxic state is not the cause, since the papilledema receded in some cases when the patient was ill and the fever and bacteremia continued, while in other cases the papilledema advanced when the patient was well enough to be ambulatory. Friesner concludes that the cranial venous sinuses are part of the dura and that the one factor which is common to all these cases is inflammation of the dura, which is the possible cause.

"Postoperative" sinus thrombosis is classified as latent and postoperative by Brunner.[34] The latent type usually occurs with acute or recurrent acute otitis,

30. Fowler, E. P., Jr.: Bacteriology of Acute Otitis Media and Its Course Under New and Old Treatment, Canad. M. A. J. **44**:372 (April) 1941.

31. Williams, H. L., and Hallberg, O. E.: Congenital Absence of the Cranial Venous Sinuses on the Right, Arch. Otolaryng. **33**:78 (Jan.) 1941.

32. Hubert, L.: Thrombosis of Lateral Sinus: Analysis of Results Obtained in One Hundred and Nineteen Cases, J. A. M. A. **117**:1409 (Oct. 25) 1941.

33. Friesner, I.: An Analysis of the Ocular Fundus Changes in Otitic Sinus Thrombosis, Ann. Otol., Rhin. & Laryng. **50**:32 (March) 1941.

34. Brunner, H.: Post-Operative Sinus Thrombosis, Arch. Otolaryng. **33**:509 (April) 1941.

when chills, increase in temperature and metastases appear after operation, especially in cases of the thrombophlebitic type of mastoiditis. The sinus usually appears normal at the original operation. This type of infection begins in venous channels connected with the sigmoid sinus. The postoperative type occurs long after mastoidectomy, especially in cases of chronic otitis. It is fulminating in its course, and there is no benefit from ligation or chemotherapy. The condition is due to incomplete removal of diseased tissue at the original operation or, more likely, to secondary infection of the wound.

Thrombophlebitis of the cavernous sinus of otitic origin is discussed by Druss,[35] who gives its incidence as from 5 to 55 per cent (rather a wide variation). The route of extension may be (1) via the inferior and/or the superior petrosal sinus from the thrombosed sigmoid sinus; (2) from the meninges secondary to meningitis, or (3) from the carotid plexus of veins, which, in turn, may be involved (*a*) directly, from a lesion of the jugular vein or the jugular bulb, (*b*) secondarily, from a suppurative process in the middle ear and the eustachian tube or (*c*) from suppuration in the apex of the petrous process. In Druss's case the infection caused by Friedländer's bacillus spread via the carotid plexus of veins, as a result of involvement of the petrous portion of the bone. The sigmoid sinus, the jugular bulb and the petrosal veins were free from disease.

Septicemia due to Friedländer's bacillus is discussed by McCall and Freeman,[36] who state that but 54 cases have been reported in the literature, with 3 recoveries. In their case, in which the septicemia occurred five days after operation, recovery followed the use of sulfanilamide and transfusions. The first operation revealed a necrotic sinus plate and an abscess along the jugular vein, producing a cervical abscess. At the secondary operation the sinus was explored, but the jugular vein was not ligated.

Kemler and Tiefenthal[37] report on spontaneous rupture of the lateral sinus in cases of otitis media (and, we presume, mastoiditis). They state that the literature shows that any of the sinuses connected with the temporal bone (usually the lateral sinus) may rupture spontaneously before or after operation on the mastoid and in cases of acute or chronic infection. The rupture occurs more often on the right side, usually about one week after operation, but may take place at any time. It is more frequently associated with the acute otitis complicating scarlet fever or with streptococcic otitis and with chronic otitis of the tuberculous or the cholesteatomatous form. In cases of acute otitis rupture is accompanied by elevated temperature and the clinical picture of sepsis. Bleeding may be external (into the external auditory meatus, nasopharynx or mastoidectomy wound) or internal (into the posterior or the middle fossa), or it may be both internal and external. Spontaneous rupture before operation has been fatal in all cases reported. Three cases are described.

PETROSITIS

One is struck by the relative paucity of articles on petrositis during the eighteen month period reviewed. Discussion of the disease is perhaps sinking back to its proper place in the literature and not overemphasized, as it certainly was for a brief period. Brown[38] discusses petrositis with contralateral symptoms

35. Druss, J. G.: Thrombophlebitis of the Cavernous Sinus of Otitic Origin, Laryngoscope **52**:115 (Feb.) 1942.

36. McCall, J. U., and Freeman, M. S.: Septicemia Due to Friedländer's Bacillus, Arch. Otolaryng. **35**:772 (May) 1942.

37. Kemler, J. I., and Tiefenthal, G.: Spontaneous Rupture of the Lateral Sinus in Otitis Media, Laryngoscope **52**:110 (Feb.) 1942.

38. Brown, E. H.: Petrositis with Contralateral Symptoms, Laryngoscope **52**:200 (March) 1942.

and explains his observations as possibly having a "toxic" basis. Hubby[39] considers the present conception of Gradenigo's syndrome and the probable mode of production of pain and ocular paralysis. The probable pathologic change is infection of the mastoid and/or the petrous process, with swelling of the lining air cells; toxins inflame the dura and give edema thereof. The dural swelling in a confined space, such as Dorello's canal, produces pressure paralysis of the sixth nerve.

OTORHINOGENIC HYDROCEPHALUS

Reeves[40] gives an excellent discussion of this condition, synonyms for which are "nonsuppurative encephalitis," aseptic meningitis, benign otitis meningitis and pseudobrain abscess. The disorder is characterized by (*a*) severe headache and elevated cerebrospinal fluid pressure (with no increase in the cell or protein content); (*b*) papilledema and occasional paralysis of the sixth nerve; (*c*) unilateral or bilateral convulsions; (*d*) a positive Babinski sign, and (*e*) nystagmus, vertigo, adiadokokinesis and deviation of the tongue. Various suggested factors in the production of this condition are: (1) interference in absorption of spinal fluid due to previous inflammatory lesions of the pacchionian bodies or perineural prolongations of the arachnoid; (2) hypersecretion of the spinal fluid; (3) closure of the interventricular foramen or the foramen of Luschka and Magendie or adhesions producing closure of the cisternae, and (4) hypertension of the cerebrospinal fluid, which may occur if the sigmoid sinuses and the jugular foramens are asymmetric and the larger side is involved, a condition which would slow down the venous circulation. Treatment is directed toward removal of the focus of infection and lowering of the cerebrospinal pressure by spinal or ventricular punctures. Chemotherapy is a valuable measure.

384 Post Street.
450 Sutter Street.
384 Post Street.

39. Hubby, L. M.: A Present Conception of Gradenigo's Syndrome, Laryngoscope **51**:1085 (Dec.) 1941.

40. Reeves, E.: Otorhinogenic Hydrocephalus, Arch. Otolaryng. **34**:920 (Nov.) 1941.

Abstracts from Current Literature

Ear

TREATMENT OF OTOMYCOSIS. MERRILL J. REEH, Ann. Otol., Rhin. & Laryng. **51**:146 (March) 1942.

All debris must be removed before treatment is started. The medicament is best applied on a wick for the first two or three days, the wick being left in place for twelve to twenty-four hours at a time. Various medicaments were used in this study, but the best results were secured from the use of metacresyl acetate and with metacresyl acetate containing 1 per cent thymol, the mixture being superior to the metacresyl acetate alone. After the wick had been in place twelve to twenty-four hours, the walls of the canal were white, dry and exfoliative, and much of the dry skin and debris could be readily removed with an applicator. If the solution proved too strong, it was diluted with olive oil. Occasionally drops of the medicament, used after the wicks had been discontinued, were diluted.

MILLER, Philadelphia.

THE RADICAL MASTOIDECTOMY: INDICATIONS, TECHNIQUE AND POST-OPERATIVE MANAGEMENT. J. D. SINGLETON, Texas State J. Med. **37**:612 (Jan.) 1942.

The anatomic relations encountered in performing the radical mastoidectomy (tympanomastoidectomy) are reviewed briefly. The indications for the operation are listed as follows: (1) intracranial complications; (2) physical signs and symptoms suggesting intracranial irritation with threatened extensions; (3) labyrinthine irritation or involvement; (4) cessation of drainage with an increase in the local and general signs and symptoms of the infection; (5) appearance of paralysis of the facial nerve, complete or partial; (6) cholesteatoma, and (7) persistent exuberant granulation tissue in the attic.

The operative procedure is simplified and accelerated by starting 1 or 2 mm. above the external auditory meatus and removing the outer two thirds of the superior and posterior walls of the canal with the cortex. At this stage it is easy to enter the antrum with a smaller gouge or curet. The operation is continued by the removal of the superior and posterior canal walls to the level of the prominence of the horizontal semicircular canal. Removal of the bridge at this stage almost completes the bony part of the operation.

During the postoperative period the operation cavity must be kept clean and excessive granulation tissue must be destroyed or removed. If the operation is well done and the postoperative care is adequate, a dry ear will be secured in most cases.

SINGLETON, Dallas, Texas.

OTITIC MENINGITIS: INDICATIONS FOR SURGICAL TREATMENT AND USE OF SULFONAMIDE COMPOUNDS. JUAN MANUEL TATO, An. de oto-rino-laring. d. Uruguay **11**:174, 1941.

Therapeusis of otitic meningitis is influenced by the following factors, among others: 1. The dynamics of inflammation, which concerns the onset, evolution and other factors that may lead, on the one hand, to regression and cure or, on the other, to chronicity or fatality. 2. The type of lesion. A lesion which is primarily vasculosanguineous, exudative or proliferative or is particularly localized to the mucosa or the submucosa is easily reached by the drug in effective concentration. On the other hand, if the lesion is either a necrosing or a proliferating process in a closed cavity, the drug is of limited value. 3. The source of infection. The infection may originate from many different otitic areas and be carried by many routes. This fact necessarily influences the various types of therapy. Meningitis may occur (*a*) by direct spread, hematogenous, tympanic or labyrinthine, or (*b*) by indirect (or secondary) propagation from a sinus thrombus or a subdural or cerebral abscess. Or meningitis may be symptomatic and occur with any of the aforementioned conditions.

The author then reports in detail a series of 11 cases, with comments on each. In the first 6 cases treatment was with one of the sulfonamide drugs alone, while in the last 5 cases both surgical intervention and chemotherapy were required. There were 9 cures and 2 fatalities (a mortality rate of 18 per cent).

Tato concludes: 1. The drug should be given in sufficient dosage and preferably by mouth, but it may also be administered intraspinally. 2. The otitic focus should be eliminated except in cases in which the pathologic process is easily regressive and the progress of the patient is favorable. On the other hand, if the situation is doubtful, it is better to operate. The author

refers again to the dynamics of inflammation in stressing the value of chemotherapy in cases in which there is ready approach (by the blood or the subarachnoid spaces) to the focus of infection.

Other measures in the treatment of this grave complication include serum therapy, lumbar and suboccipital puncture, blood transfusion and intravenous injection of dextrose and a hypertonic saline solution.

PERSKY, Philadelphia.

PETROSITIS. A. TELLO and J. S. RIESCO, Rev. otorrinolaring. **1**:69 (March) 1942.

Tello and Riesco describe 2 cases of petrositis in which the operation was performed by the carotid canal route. They then discuss the clinical phases of this complication, its progress along the various cell routes, the symptomatology and the many difficulties in diagnosis. They give a detailed discussion of the anatomic relations, graphically describing the arrangements of the various cell groups in the region of the labyrinth and the carotid apex. As an index to the various operative procedures, the authors accept Ramadier's classification, according to which petrositis may be subdivided into (*a*) posterior perilabyrinthitis, in which the superior, inferior and internal posterior cells are involved, and (*b*) anterior perilabyrinthitis, in which the involvement may be supracochlear, subcochlear, precochlear or apical. For the posterior type complete radical mastoidectomy is sufficient. When the supracochlear cells are involved they advocate Myerson's technic—a thorough, radical mastoidectomy with exenteration of the cells of the antrum, the tympanum and the subdural tissue throughout the course of the facial nerve. When the precochlear cells are involved, the technic of Kopetzky and Almour is recommended. When the apex is involved there are a number of procedures, such as those of Frenchner, Almour, Ramadier and Lempert and the extrapetrosal route of Eagleton. The authors favor the Lempert approach to the carotid region and describe his technic in detail. One patient recovered, while the other succumbed as a result of generalized septicemia.

PERSKY, Philadelphia.

PATHOLOGY AND TREATMENT OF OTOGENIC MENINGITIS IN CHILDHOOD. E. JENTS, Monatschr. f. Ohrenh. **75**:225 (May) 1941.

The author had opportunity to observe 8 children with otogenic meningitis, 6 of whom survived the disease. The ages ranged from 14 months to 10 years. In 6 patients the primary disease was scarlet fever or some other disease of childhood, in the course of which otitis developed, followed by meningitis. In 2 patients simple acute otitis was followed by meningitis. The meningitis developed in different stages of the otitis media, after four to thirty-one days. No other intracranial complication was observed in any instance. The diagnosis of otogenic meningitis is sometimes difficult to make when the child is suffering from some other disease which might simulate meningitic symptoms, such as scarlet fever or measles. Therefore early spinal puncture as a diagnostic means is advisable. Acute infection of the meninges produces polynucleosis, while in a later stage mononucleosis is present. This increased cell count, together with the clinical symptoms, is of great importance as an indication for operation. An average of the cell counts should be taken, as they vary with the sample of spinal fluid examined. In 5 cases the culture was found to be positive; in 4 instances Streptococcus pyogenes haemolyticus, and in 1 case Staphylococcus albus, was found. In both the fatal cases streptococci were present. It might be difficult to establish the picture of independent meningitis, but clinical symptoms, together with the proper changes in the spinal fluid, will support the diagnosis and indicate surgical intervention. It is essential to eliminate the focus of infection and to relieve intracranial pressure. Various operative methods can be employed. The author usually performed complete mastoidectomy, with exposure of the posterior and middle fossae. In addition, the connecting "bridge" is removed, a large exposure being thereby achieved. Jents refuses to perform an atticoantrotomy, as has been advised by Neumann, and is opposed to a radical mastoid operation. The second measure is necessary to combat the meningitis proper. This is carried out by reducing the increased pressure on the brain by means of daily spinal taps, with the removal of 30 to 80 cc. of fluid and the introduction of 20 to 30 cc. of air. Furthermore, chemotherapy in the form of azosulfamide (disodium 4-sulfamidophenyl-2',-azo-7'-acetylamino-1'-hydroxynaphthalene-3', 6'-disulfonate) and cylotropin (a preparation of methenamine and sodium salicylate) has been employed by the author. Prontosil was administered intrathecally and intramuscularly, as well as by mouth. The general resistance has to be built up and the heart supported. Repeated blood transfusions seem to be of great therapeutic value. The author feels that good results are achieved when these conditions are borne in mind and otitis media of childhood is not regarded as a minor ailment but the child is hospitalized in due time, so that early operation can be performed.

LEDERER, Chicago.

RELATION OF IRRITATION OF THE LABYRINTH TO THE BLOOD SUGAR LEVEL. J. BERBERICH, Pract. oto-rhino-laryng. **3**:342, 1941.

In a series of experiments with animals, Berberich noted that when the labyrinth had been stimulated, either by the rotatory or by the caloric method, there was an increase in the blood sugar, which might persist for a half-hour. The increase may be as great as 20 per cent within the first five minutes after stimulation. A similar result was also obtained by stimulation with both chloroform and ether. Berberich's explanation is that during the stimulation epinephrine is poured into the circulation in increased amounts and more glycogen is mobilized in the liver. This is probably in accordance with the observation that there is a slight increase in the blood pressure during the test. The author offers another explanation, namely, that by way of the vagus nerves impulses from the labyrinth go to the central portion of the brain and then to the floor of the fourth ventricle, where the sugar center is located. The afferent tracts of the nucleus vestibularis are connected with the vagus nerve through the nucleus triangularis.

PERSKY, Philadelphia.

Pharynx

CHRONIC GRANULAR PHARYNGITIS. RAYMOND H. MARCOTTE, Ann. Otol., Rhin. & Laryng. **51**:406 (June) 1942.

Postnasal discharge is only one of the causes of chronic pharyngitis. Granular pharyngitis may appear after tonsillectomy, apparently being an attempt to replace removed tissue. It develops in mouth breathers, probably owing to dryness resulting from the absorption of pharyngeal moisture by inspired air, with production of capillary dilatation and local inflammation. Dust and irritating gases in industry may produce it. Certain pharynxes are irritated by tobacco smoking. Some general systemic diseases, such as constipation, rheumatism, hepatic cirrhosis and cardiac disease, may predispose to chronic pharyngitis, as a result either of the accompanying toxicity or of circulatory disturbances. Dietary errors, faulty metabolism or endocrine dyscrasias may cause pharyngeal changes. Allergic pharyngitis is seen, but granulations rarely appear unless complications exist.

In diagnosis of granular pharyngitis, a thorough examination must be made to eliminate the purely catarrhal type due to sinusitis, and the postnasal area, the larynx and even the pulmonary system should not be overlooked. Various treatments have been advised: simple irrigation of the nose and pharynx with saline solution, vaccine therapy, irradiation with a quartz-mercury vapor lamp, roentgen irradiation, electrocoagulation, surgical removal, application of 50 per cent silver nitrate and internal medication with iodine. The author prefers the use of silver nitrate or electrocoagulation, together with administration of iodine internally. He feels that disturbance of the thyroid has no bearing on the condition.

M. V. MILLER, Philadelphia.

ANALYSIS OF SEVEN HUNDRED AND SIXTY-FIVE TONSILLECTOMIES BY THE SLUDER TECHNIC. RENE ESPINOSA, Rev. otorrinolaring. **1**:87 (March) 1942.

Espinosa advocates the Sluder procedure in all cases of tonsillectomy. In his series of 765 cases there were only 3 instances of hemorrhage. He states that there is often some residual tonsillar tissue, usually at the inferior pole, that must be removed by a snare wire.

PERSKY, Philadelphia.

Nose

LACERATION OF THE MUCOUS MEMBRANE AND HEMATOMA OF THE NASAL SINUSES IN AVIATORS. A. HERMANN, Ztschr. f. Hals-, Nasen- u. Ohrenh. **47**:103 (Dec.) 1940.

Hermann presents a series of 4 cases of spontaneous hematoma of the nasal sinuses occurring in aviators. The pathologic change was localized to the frontal sinus in 3 cases and to the maxillary sinus in the fourth. The histories were similar in all instances—a sudden attack of severe pain associated with a sensation of tearing of tissue in the involved area, occurring during a flight. In 3 of the cases there was a history of recent infection of the upper respiratory tract. Rhinoscopic examination was inconclusive, but roentgenographic studies revealed a well defined shadow in the involved sinus. This shadow was diagnosed as either a mucocele or a pyocele. However, an operation performed in 1 of the cases in which the frontal sinus was involved and another in which the maxillary sinus was implicated revealed laceration of the mucous membrane, with separation from the underlying bone, and a submucous hematoma. Uneventful recovery followed the operative procedure. In the other 2 cases expectant treatment, supplemented by short wave therapy, was employed and

in both recovery was spontaneous. In conclusion, the author stresses (1) the importance of the roentgenographic signs as noted, particularly in patients with well developed frontal sinuses, (2) the danger of a recent infection of the upper respiratory tract among aviators and (3) the importance of local expectant treatment, particularly directed to the control of the intranasal condition, and of short wave and roentgen therapy. PERSKY, Philadelphia.

OSTEOMYELITIS OF THE FRONTAL BONE. HEINZ G. A. BAYER, Ztschr. f. Hals-, Nasen- u. Ohrenh. 47:202 (Dec.) 1940.

Bayer presents an exhaustive survey of osteomyelitis of the frontal bone. In the first section he discusses the pneumatization of the frontal sinus in lower animals. He observed that monkeys (throughout all groups) showed a development of the ethmoid and frontal sinuses similar to that in man. Bayer then discusses the embryonic development of the frontal sinus in man. There seems to be a primary and a secondary pneumatization. The secondary pneumatization begins about the third to the fourth year and ends in the third to the fourth decade of life. To explain the development of the sinus, he presents two theories: the one, that the anterior ethmoid cells encroach on the frontal bone and resorption of the bone results, and the second, that intranasal air pressure is exerted on the frontal bone and its anterior and posterior walls recede. Various factors influence abnormal development of the sinus. Among these are an intranasal pathologic process, congenital syphilis, familial predisposition, ozena and constitutional factors. The author also observes that (1) women usually have smaller sinuses than men; (2) the left sinus is usually larger than the right, and (3) the septum of the sinus is usually deviated to the right.

Bayer then elaborates on the arterial, venous and lymphatic supply of the sinus. He stresses the importance of the communicating vessels, as well as the interosseous (diploic) sinuses and veins, which were described by Breschet. The lymphatic circulation also plays an important role in osteomyelitis.

In the second section of this paper, Bayer discusses the roentgen diagnosis of disease of the frontal bone. Many technics are employed. These include the use of the usual flat plate in various planes, stereoscopic films in different axes and tomography. These procedures are necessary since certain defects, either due to size or location, may be visualized by one technic and yet be found to be entirely absent with another procedure. Osteomyelitis can be diagnosed only when either a productive or a resorptive process is present in the involved bone, but an acute exudative condition cannot be diagnosed with any degree of accuracy. An osteomyelitic process may be present for one week to several months without any clearcut roentgenographic evidence of the lesion.

In the third section, Bayer discusses first the causation of osteomyelitis. The disease may be traumatic, may follow inflammatory disease in the pneumatic spaces of the skull, may have a metastatic hematogenous origin or may be primary. The incidence is difficult to determine, but he assumes that it constituted about 1 to 2 per cent of his cases of frontal sinusitis, that the disease is more common in males and is most frequent between the ages of 10 and 30.

The author then discusses the pathologic process in the bone marrow, the periosteum, the suture lines and the bony tissue. His observations indicate that the osteomyelitic process usually begins in the bone marrow of the zygomatic region and spreads from this site. While he does not deny the possibility of extension by way of the diploic vessels, directly through the sinus wall, or of a periosteal or an extraperiosteal spread, he was able to determine such a route only in a single case.

He then discusses the clinical and diagnostic features and concludes that there are few symptoms, inclusive of the blood picture, that can predict oncoming osteomyelitis. There is often great difficulty in interpreting the "doughy" swelling over the frontal bone. However, in the presence of a more or less profuse nasal discharge, frontal pain, "doughy" swelling, headache, edema of the lids and chemosis, nausea and vomiting, high fever and pronounced leukocytosis, a tentative diagnosis of osteomyelitis should be made.

The prognosis is influenced by the associated complications. It is usually grave. Bayer's mortality rate was 52 per cent. In his 25 proved cases, there were 94 complications, with 13 fatalities. Of the 13 deaths, 9 occurred after an acute, stormy course, while 4 followed a mild, insidious onset. The complications were extradural abscess, leptomeningitis, brain abscess, venous thrombosis, subperiosteal abscess, orbital disease and metastatic embolism.

The treatment is both local and general. The operative procedure should be as radical as necessary and as early as possible. Neither the local macroscopic appearance nor the roentgenographic evidence is of real assistance in determining the extent of the pathologic change. The bone should be removed at least for 1 inch (2.5 cm.) beyond the gross demarcation

between diseased and apparently healthy bone. This operative procedure should be supplemented by systemic therapy, such as serum therapy and chemotherapy, transfusions, a suitable dietary regimen and other supportive measures.

It is noted that therapy with one of the sulfonamide compounds was employed with only 2 patients, each of whom recovered, although 12 other patients were reported on recently enough to have the benefit of this type of medication. Four of the last 12 patients died.

PERSKY, Philadelphia.

Miscellaneous

SUPPORTIVE THERAPY IN BRONCHIAL ASTHMA AND VASOMOTOR RHINITIS. JOSEPH S. STOVIN, New York State J. Med. **41**:463 (March 1) 1941.

Citing the report of the Asthma Research Council after five years of investigation, Stovin states that the reasons for dissatisfaction are to be found in too much dependence on desensitization and not enough on body chemistry. There may be an unrecognized imbalance, for example, between the cations of sodium and those of potassium. In contrast to the diminishing effectiveness of preparations of epinephrine is the sustained, and often increasing, effectiveness of supportive therapy—administration of potassium and restriction of sodium chloride.

"The use of diet rich in protein and acid ash and low in sodium chloride, combined with the administration of potassium iodide and bicarbonate, has proved effective. . . . A similar diet combined with the administration of the chloride salt of potassium has given excellent results in the general treatment of vasomotor rhinitis with no, or only slight, associated asthma."

VOORHEES, New York.

HEMORRHAGES RESULTING FROM EROSION OF VESSELS IN THE NECK BY MALIGNANT TUMORS. P. REHÁK, Monatschr. f. Ohrenh. **75**:76 (Feb.) 1941.

The material of the Budapest clinic permitted the study of hemorrhages caused by eroded vessels. After total laryngectomy, bleeding was noted as originating from local recurrences of hemorrhage around the hyoid bone and breaking through against the pharynx or the skin or both. At the side of the neck hemorrhage usually arises from metastatic glands and perforates the skin. Finally, bleeding occurs around the tracheostomy opening. In patients treated by irradiation a discolored skin will indicate the area where bleeding is likely to be found, usually in the places mentioned. Malignant growths other than those of the larynx perforate the skin near the sternocleidomastoid muscle or the hypopharynx. Ligation of vessels is sometimes difficult if the tissue is infiltrated, and not even ligation of the carotid artery will help, as sometimes the thyrocervical trunk is responsible. Deep sutures knotted over a tampon will occasionally stop such hemorrhages.

LEDERER, Chicago.

INFLUENCE OF TONSILLECTOMY ON THE BLOOD PICTURE. E. URBANTSCHITSCH, Monatschr. f. Ohrenh. **75**:397 (July) 1941.

Within two hours after tonsillectomy leukocytosis appears, with a definite shift to the left, usually lasting for three days. Increase in young forms and rod forms of granulocytes and a decrease in lymphocytes usually follow. Destruction of the granular elements and unripe forms have also been observed. The studies were carried out on 41 patients.

LEDERER, Chicago.

PRACTICAL ALLERGIC TESTS. H. A. E. VAN DISHOECK, Pract. oto-rhino-laryng. **3**:321, 1941.

Van Dishoeck discusses the difficulties encountered in the routine testing of allergic patients. In order to simplify the usual cumbersome and time-consuming procedure, he advocates, first, that the cutaneous method with the use of the dry allergen be employed. This allergen is cheap and does not deteriorate. Second, he recommends that a rational selection of allergens be made. He used a group of 20 allergens which represented the normal surroundings and foods in a specific locality. This major group was then divided into 17 subgroups, and with the latter he was able to limit his preliminary tests to 17 cutaneous scratches. However, for a differential diagnosis he employed 26 of the more important allergens of any one of the aforementioned groups if it gave a positive reaction.

Van Dishoeck then evaluates the responses of each group of allergens and expresses the belief that cutaneous tests are important from a diagnostic standpoint and as a guide to therapeusis. On the other hand, they are of limited significance in the presence of nonspecific allergy.

PERSKY, Philadelphia.

DIAGNOSIS AND TREATMENT OF ABSCESS OF THE BRAIN: A CLINICAL STUDY. NILS LUNDBERG, Acta oto-laryng. **29**:36, 1941.

Abscess of the brain develops in a manner similar to abscess in other parts of the body, but an added factor, increased intracranial pressure, must be considered. For a successful outcome it is not sufficient to overcome the infection, but this must be done before the increased intracranial pressure kills the patient.

Various processes cause changes in the intracranial pressure, namely, expanding lesions, hemorrhage, disturbances in formation and circulation of the cerebrospinal fluid, edema of the brain caused by inflammation of and damage to the brain tissue and changes in the venous pressure and in the osmotic condition of the blood.

In the case of an expanding lesion with a sudden rise of pressure there not infrequently develops a so-called pressure cone, in which the brain stem is pushed down into the foramen magnum. The symptoms of this complication are interrupted breathing, nuchal pain and rigidity and a forced position of the head. A lumbar puncture can produce the same picture and is contraindicated when an expanding lesion is suspected.

The clinical picture of increased intracranial pressure consists of somnolence, headache and symptoms of the "pressure cone." Bradycardia and changes in the breathing or the blood pressure are of less importance in determining the presence of increased intracranial pressure, particularly in cases of brain abscess, in which the infection itself clouds the picture.

Roentgenography.—In modern brain surgery the preoperative diagnosis is almost 100 per cent roentgenologic. The roentgenographic examination is preceded by encephalographic, ventriculographic and angiographic tests, and the diagnosis is made on the basis of the deformities and dislocations of the arachnoid spaces visualized by these means. Ventriculography is of more value than encephalography because with increasing intracranial tension the cisterna magna is often closed to the passage of fluid or air. In the presence of an expanding lesion there is a small amount of risk in performing a ventriculographic examination, and it should be done only if one is prepared to operate immediately. The risk of ventriculography is somewhat greater in the case of an abscess than in that of a tumor of the brain because the immediate operation does not relieve the intracranial tension as rapily in the former as in the latter. Therefore, an encephalogram should be made instead of a ventriculogram when possible.

To obtain a cerebral angiogram 8 to 10 cc. of thorium dioxide is rapidly injected into the internal carotid artery and a roentgenogram is rapidly taken. Or the contrast medium can be injected into the venous septum. Angiography does not carry the same risk of increasing the intracranial pressure as does ventriculography, but it does increase the tendency to hemorrhage, and, inasmuch as the thorium is retained throughout life in reticuloendothelial cells of the liver and spleen and is radioactive the substance may have some carcinogenic effect. Furthermore, it is of no value in the diagnosis of lesions in the posterior fossa, which is not supplied by the internal carotid artery.

Diagnostic Puncture.—Since the advent of the roentgenologic diagnosis the diagnostic puncture has lost much of its importance. It should be reserved for the exact localization of abscesses of the brain the general position of which is already known.

Surgical Treatment.—The classic work in the surgical management of brain abscess is that of Macewen, who made it his rule to incise and drain as soon as the diagnosis was made and who reported 19 cases in which this treatment was carried out, with a mortality of only 10 per cent. In cases of rhinologic and otogenic abscesses he first operated on the suppurative focus, but never used a gouge because of the danger of damaging the brain tissue and spreading the abscess. If he encountered a fistula he widened it, but because this gave insufficient drainage he also trephined as close to the abscess as possible, cut the dura and punctured the abscess with a dull needle. He then incised along the needle and excised a part of the cortex. He deprecated drainage of an early abscess and sewed the dura and the soft parts with a primary suture. In cases of chronic abscess with capsule formation he drained with decalcified chicken bone or rubber tubes, which he sewed to the skin and kept in situ for three weeks. In most of his cases the abscess was chronic with a well formed capsule, and therein may have lain his phenomenal success.

In contradistinction to Macewen's closed drainage method, Cushing, and later Horrax, were exponents of the open drainage method, in which a relatively large craniotomy (4 to 5 cm.) was made, the brain tissue over the abscess excised, the abscess membrane incised and stitched back and the abscess cavity filled with an air-distended rubber sac. The pressure of the brain gradually pushes the abscess floor outward, so that by and by it comes to lie on

the same level as the outer surface of the brain. Further prolapse is prevented by lumbar puncture. Horrax treated 9 chronic brain abscesses by this method, with 1 death; King, 17 abscesses with 3 deaths, and Bucy, 17 abscesses with a mortality rate of 29 per cent.

Dandy objects to all forms of drainage and contents himself with puncture on the ground that susceptible brain tissue in the neighborhood of the abscess suffers damage by the incision, drainage tubes or gauze wicks, which injury raises the pressure in the brain, causing herniation and increased irritation of the brain tissue. In certain cases he combined the puncture treatment with trepanation. He expressed the belief that by his method the infection can be controlled and the abscess cured before the patient dies of increased intracranial tension.

Because of diagnostic errors, neurosurgeons have for a long time occasionally treated an abscess of the brain by extirpation through an osteoplastic flap. Vincent began to use this method purposefully and reported 10 cases in which it was successful, but neglected to give his mortality figures. He extirpated the chronic abscess with a well formed capsule directly. He treated the early abscess by puncture and decompression in the form of a flap over the abscess until the capsule was resistant enough, after which it was excised. Horrax and Kahn also treated abscesses successfully by this method. The four principles in the surgical treatment of abscess of the brain are therefore closed drainage, open drainage, puncture and excision. Each one of these methods has its place, and one must individualize the treatment of every such abscess.

Time for Operation.—As important as a proper choice of operation is the choice of a proper time for the operation. There is unanimity of opinion that for a successful result the abscess must have a resistant capsule. Grant operated on 19 abscesses without a capsule with 100 per cent mortality and on 30 abscesses with a capsule with only 33 per cent mortality. According to Penfield, the abscess membrane begins to form in the first week, but only regularly offers resistance to the puncture needle after three to four weeks.

According to Grant, one can expect a resistant membrane only after four to six weeks. He expressed the opinion that six weeks after the abscess forms is the ideal time for operation. An estimate as to the resistance of the abscess can be obtained with the puncture needle. Waiting also offers the advantage of increased immunity and lessened intracranial pressure. In waiting one naturally must be guided by the brain pressure and by neurologic evidence of extension of the process. In addition to the actual operation on the abscess, the most important adjuncts of abscess therapy are the treatment of the primary focus, the relief of intracranial pressure and chemotherapy.

Treatment of Primary Focus.—Under ordinary circumstances the primary focus should be treated radically before the abscess is operated on. An exception to this policy is made when there is a decided increase in intracranial pressure. Whether the abscess should be operated on from the same operative field as the primary focus can be argued. Lundberg thinks that it should, provided there is a demonstrable fistula. Otherwise he recommends operating on the abscess through a clean field.

Treatment of Intracranial Tension.—When an acute rise in pressure occurs, one makes use of intravenous medication with hypertonic solutions. While ventricular puncture causes the pressure to drop momentarily, it produces an increase later and should be avoided.

When a chronic increase in brain pressure is present, the treatment consists in dehydration by means of magnesium sulfate, given by mouth, limitation of fluid intake and, possibly, diuretics. Of more importance is the avoidance of everything which may increase the pressure, such as constipation, vomiting, ether narcosis and enemas.

Chemotherapy.—The advent of sulfanilamide and sulfapyridine and their derivatives will probably revise former indications for treatment and therapeutic methods. These drugs offer the possibility of limiting the local cerebral infection before the increasing pressure becomes deleterious and thereby improve the likelihood of a favorable waiting period. Their use makes for less danger of postoperative meningitis and increases the possibility of radical, and even early operative, intervention.

Lundberg gives the histories in 4 cases, of which recovery resulted in 3 and death in 1, a mortality rate of 33.3 per cent.

GROVE, Milwaukee.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OTOLARYNGOLOGY, AND PHILADELPHIA LARYNGOLOGICAL SOCIETY

KARL M. HOUSER, M.D., *Chairman*

LOUIS J. BURNS, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

THOMAS F. FURLONG JR., M.D., *Secretary*

Regular Meeting, Nov. 18, 1942

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Orbital Cellulitis. DR. HORACE JAMES WILLIAMS.

Patients with orbital swelling (cellulitis) are likely to be seen at irregular intervals by every otolaryngologist. Knowledge of its anatomic, etiologic, pathologic and clinical features is essential to proper management. Improper conduct of a case may lead to an untimely or inexpedient operation, with impairment or loss of vision, or even to death from meningitis or thrombosis of the cavernous sinus. The present concept of the cause of orbital cellulitis is comparatively new. Such terms as "primary" and "idiopathic" are seldom used in connection with this pathologic condition, while "thrombophlebitis" and "secondary to paranasal sinusitis" have become common.

The most common place in which an abscess may form is between the superior and the mesial bony wall of the orbit and the periorbita. An abscess in this location loosens the periorbita, which strips away easily, and points in the upper lid or on the side of the nose, with consequent displacement of the eye, and at times exophthalmos.

With scarlet fever orbital swelling may assume alarming proportions, but conservative treatment is usually successful. In general children with sinusitis are susceptible to edema of the orbital contents. Resolution usually follows adequate treatment.

In a discussion of orbital swelling one must consider three stages of the disease: First, and most frequent, is simple edema of the lids, which is infiltration of the part with serum. This edema resembles that of the lids during acute nephritis. The action of the toxins from the infected sinus increases the permeability of the capillaries, and fluid pours out into the intracellular spaces. If therapy is instituted promptly, and the sinus producing this lesion responds to the treatment, the edema subsides in twenty-four to forty-eight hours.

The second stage is orbital cellulitis, which is acute diffuse inflammation of all or part of the contents of the orbit. This process is characterized by swelling of the lids and conjunctiva. Pain and fever are usually present. The pain may be severe and the fever high.

The third stage is orbital abscess, which is a localized suppurative lesion, usually subperiosteal. Exophthalmos may be present, depending on the location of the abscess. The symptoms are much the same as those of orbital cellulitis, but more severe. Pointing in the upper or the lower lid may take place. Often it is difficult to differentiate between orbital cellulitis and orbital abscess.

Orbital swelling of nasal origin has to be differentiated from orbital swelling resulting from other causes, such as tumor, trauma, foreign bodies or thrombosis of the cavernous sinus.

When definite protrusion of the eyeball is associated with orbital cellulitis, it must be differentiated from thrombosis of the cavernous sinus. In cases of the latter condition the symptoms are more severe. The temperature is high and fluctuating and the elevation is usually associated with chills. Edema of one side is soon followed by edema of the other. A history of infection of the upper lip, the nose or a tooth is occasionally obtained.

In adults infection of the frontal sinus is the most frequent cause of orbital cellulitis; in children the ethmoid cells are usually responsible, and in very young children the maxillary sinus is not an infrequent offender. Infection arising in the frontal sinus enters the orbit from the

floor of the frontal sinus, just to the inner side of the supraorbital notch. From the ethmoid cells the rupture occurs just posterior to the posterior lacrimal crest. A diseased maxillary sinus usually produces edema of the lower lid alone.

If the edema increases, instead of responding to conservative treatment, such as shrinkage of the nasal mucosa with a solution containing 1 grain (0.065 Gm.) of cocaine hydrochloride and 5 grains (0.325 Gm.) of ephedrine sulfate in 1 ounce (29.5 cc.) of physiologic solution of sodium chloride several times a day, thorough cleansing of the nasal cavity with a warm saline solution or by suction with a soft rubber catheter, administration of a sulfonamide compound and the application of cold compresses every hour for twenty minutes, which is of great value; if the pain and tenderness become worse, with a further increase in temperature, and the swelling changes from simple edema to orbital cellulitis, the time has arrived when a decision must be made. Should conservative treatment be continued; should an intranasal opening be made into the ethmoid cells or the frontal or maxillary sinus, or should the intranasal procedure be carried out in connection with external drainage?

A roentgenogram is of inestimable value. It has been my experience that a combined external and internal operation produces better results than simple removal of the anterior third of the middle turbinate and the opening of the anterior ethmoid cells. "Conservatism is the rule, but those who have voiced an ultraconservative attitude have probably experienced in their own hands or in those of their colleagues unfortunate sequelae."

Radical operation is not attempted during the acute stage. If the maxillary sinus is involved, irrigation is usually sufficient, but if the edema of the lower lid continues, a permanent opening should be made under the middle turbinate. As most cases of orbital edema from maxillary sinusitis occur in infants, puncture is usually sufficient. The after-treatment consists in keeping the wound clean and the nasal cavity free of secretion.

So far in my experience, osteomyelitis has developed in but 1 case, that of a man with chronic pansinusitis of many years' duration. He minimized his symptoms to such an extent that operation was postponed until osteomyelitis had developed. The termination was fatal.

Conclusions.—1. Conservatism in treatment should be the rule, but if fever and pain persist, with some evidence of meningeal irritation, even though edema is not great, the sinus involved should be drained.

2. Thrombosis of the cavernous sinus is the condition most likely to be confused in diagnosis.

3. The principal risk in external operation is diplopia resulting from interference with the action of the superior oblique muscle. When this complication occurs, function returns in a few weeks.

4. Fear of scarring should not be a reason for failure to operate. After a few months the scarring is negligible. The wound closes easily even if it has been left open.

5. Operation is preferable to development of a complication. Complications are difficult to manage and may be fatal.

6. In about 200 cases, I have never seen an abscess develop between the periorbita and Tenon's capsule.

DISCUSSION

DR. SAMUEL R. SKILLERN JR.: Two cardinal principles should govern all efforts to cure this condition, namely, improvement of metabolism and establishment of drainage, drainage both of the diseased cell into the nose and of the nose.

Not only does a child with obstructed breathing harbor disease-producing germs and irritating matter within the nose, but the obstruction deprives him of nature's means of clearing the various cells and sinuses, namely, blowing the nose, sniffing and hawking. To know how important are these forces, puncture an antrum with a cannula, fill the cannula with a colored fluid and ask the patient to sniff and hawk. The fluid in the cannula disappears immediately, to appear in the nose and throat. A process of blowing produces the opposite effect; that is, the fluid is blown from the cannula. Therefore, with a child suffering from any form of sinusitis, one should see that all adenoid tissue is removed from the nasopharynx. The nose should be cleared of secretion, and thereby the sinuses will be much assisted in emptying themselves.

In many cases a chronic condition will recover under the aforementioned treatment if it is persisted in somewhat longer than is usually required for the acute stage. In others improvement refuses to progress beyond a certain point. In such cases and in those of fulminating conditions in which danger symptoms develop early surgical intervention is required. Here is demanded not technic alone but judgment.

With respect to children, my feeling as to operation on the various sinuses is as follows:

Frontal Sinus.—No attempt should be made to open the frontal sinus intranasally in children under 12 years of age. If the frontal sinus is diseased, the ethmoid cells are probably diseased coincidentally, and a complete Killian operation should be done.

Ethmoid Cells.—The ethmoid cells are frequently diseased independently, at least of the frontal sinus. The safest and most promising procedure is the second part of the Killian operation, performed through an external incision. Exenteration of the ethmoid cells intranasally with the child under general anesthesia is dangerous.

Sphenoid Sinus.—When exenterating the ethmoid cells, one should, if there is any question of disease of the sphenoid sinus, enter this sinus at the same time. The walls of the sphenoid sinus cannot be curetted without danger.

Antrum.—Probably the antrum is the sinus which most frequently demands operation in childhood. If the permanent teeth have erupted, the problem is simple. If the sinus has refused to clear up after an opening into the sinus has been made high under the inferior turbinate, through which considerable washing and medication have been carried out, the Caldwell-Luc operation should be performed. With younger children, whose second teeth have not descended, one must exercise judgment in determining whether to enter the antrum through the canine fossa, at the expense of one or two teeth, or to delay the radical operation until such time as the second teeth shall have erupted. I have never observed bad results from the sacrifice of a tooth. I have never seen lack of facial development or overproduction of osseous tissues from such a procedure, except in the case of a malignant growth. That these direful results follow radical operation carried out through the canine fossa in children I believe to be argued from academic premises.

Response of the Labyrinthine Apparatus to Electrical Stimulation. Dr. E. A. Spiegel and Dr. N. P. Scala (by invitation).

This article will appear in full in a later issue of the Archives.

DISCUSSION

Dr. George W. Mackenzie: It is a privilege to discuss a paper presented by Dr. Spiegel and Dr. Scala, for both of whom I hold the highest esteem.

Bárány, in 1907, presented a brief outline of the improved method of examination for galvanic nystagmus of each ear separately and cited Neumann's experiment of galvanization of the eighth nerve after labyrinthectomy. He concluded that the galvanic method of examination was of no practical value.

Most of what I have to say will be limited to monaural galvanic reactions in health and in disease. All tests I have made have been monaural. An assistant aids with the test and moves the switch controlling the strength of current, calling out the number of milliamperes as the current is increased, while the observer notes the nystagmic movements of the eyes. Care is taken that the patient at all times looks straight ahead in order to eliminate that important variable, physiologic nystagmus. Any deviation of the eyes from one side to the other influences the intensity of the nystagmus in favor of the direction in which the patient is looking.

In my examinations of deaf-mutes in Vienna, I found that in cases of disease of the middle ear and labyrinth the reactions were positive, whereas in cases of meningitis no reaction was elicited, even with currents as high as 14 milliamperes.

Although I point out the greater applicability and value of the galvanic tests over other tests, do not misunderstand me as condemning the other methods. On the contrary, I find them of considerable value. The galvanic test has served me faithfully in differentiating bilateral neurolabyrinthitis syphilitica, with absence of galvanic reactions, and bilateral labyrinthitis syphilitica tarda, in which the galvanic reactions are normal.

Many things could be said in commenting on the excellent paper by Drs. Spiegel and Scala, but lack of experimental research on animals forbids me to indulge. In closing, I wish to acknowledge the value of animal experimentation in providing essential preliminary groundwork for the clinical study of human beings.

Dr. Benjamin H. Shuster: Dr. Spiegel's observations on the reactions of the labyrinth to stimulation with electric currents should be of clinical value.

His first conclusion is that the faradic current stimulates labyrinthine responses in the same way as does the galvanic current provided the faradic frequencies are very low. This disproves the idea that the galvanic current alone is a specific agent in stimulation.

His other point, that section of the eighth nerve medial to Scarpa's ganglion causes degeneration of that nerve, with consequent absence of responses to electrical stimulation, does not coincide with the fact that after labyrinthitis, which is a lesion peripheral to Scarpa's ganglion, there exists some response at first but the reactions disappear. This seems to indicate that degeneration takes place even when the lesion is peripheral to the ganglion.

Dr. Julius Winston: Unfortunately, my discussion, too, must be confined to the clinical side, rather than to animal experimentation.

Electrical stimulation, save for its ability to differentiate between a lesion in the semicircular canals and one of the nerve proper, has little clinical application. When one is called on to apply the vestibular tests in the diagnosis of intracranial lesions, as one so often is, one finds that frequently with caloric stimulation the vertical canals are not responsive while the horizontal canal reacts normally. It has been pointed out that the electric stimulus stimulates the end organs in all the semicircular canals, and so does not reveal absence of function of the vertical canals.

The question of susceptibility to vestibular stimulation, that is, shock reaction, is important clinically. Inasmuch as the electric stimulus is applied for a brief period, shock reaction practically never occurs.

Furthermore, when there is a partial or total loss of vestibular function, the galvanic stimulus must be increased in intensity above the normal 4 to 6 milliamperes, so that, contrary to Dr. MacKenzie's statement, the electric stimulus is infinitely more painful than the caloric stimulus.

Dr. E. A. Spiegel: I wish to express my sincere appreciation to all the discussers for adding their important clinical experiences to our experimental observations. My own impression is that the clinical usefulness of the galvanic test is limited chiefly to the differential diagnosis of labyrinthine and retrólabyrinthine lesions. In using galvanic stimulation for this differential diagnosis, one has to bear in mind an important point, which also answers Dr. Shuster's question. If several weeks elapse after extirpation of the peripheral labyrinth, destruction of the peripheral endings of the vestibular nerve induces secondary degeneration of the cells of the ganglion vestibulare Scarpae and, consequently, the vestibular nerve loses its excitability to galvanic stimuli, so that the primary lesion of the labyrinth is followed by the picture of retrolabyrinthine disease due to the secondary degeneration of the neuron of the vestibular nerve. Dr. Mackenzie's interesting observations on monopolar stimulation in cases of labyrinthine disease can easily be understood if one bears in mind that elimination or hypofunction of one labyrinth, e. g., the left, will induce an imbalance between the left and the right vestibular nucleus and a latent tendency to nystagmus toward the normal side. Consequently, the threshold for stimuli inducing nystagmus to the normal side (e. g., cathodal stimulation of the right labyrinth) will be lowered, and the threshold for stimuli inducing nystagmus to the side of the diseased labyrinth (e. g., anodal stimulation of the right side) will be increased. I can touch only briefly here on the problem of the so-called lack of excitability of the vertical canals in cases of increased intracranial pressure. This symptom, discovered by Eagleton, is of high diagnostic importance. In interpreting it, one has, however, to bear in mind that in these cases the vertical canals are still excitable for rotational stimuli, as was first emphasized by the French author Hautant. The caloric stimulus is a rather weak stimulus for the vertical canals; thus they easily fail to respond to calorization if the reactivity of the labyrinth is impaired, while the stronger rotational stimuli may still reveal excitabilty.

Lymphoepithelioma of the Nasopharynx: Report of a Case. Dr. A. H. Fersky.

This article will appear in full in a later issue of the Archives.

DISCUSSION

Dr. William Hewson: In discussing this case, I wish to stress two points: First, tumors in the nasopharynx are difficult to diagnose because of their hidden location, and therefore present a challenge, as Dr. Persky indicated, for more thorough examination with the nasopharyngoscope and the nasopharyngeal mirror. Both methods should be used in doubtful cases, because each is insufficient in itself. In doubtful situations the nasopharyngoscope is of particular use in examining the vault of the nasopharynx and the posterior choanae, while the nasopharyngeal mirror is best for lower lesions, particularly those involving the posterior wall of the nasopharynx.

Second, tumors of the nasopharynx are often obscure or difficult to diagnose because of mixed histologic or embryonic elements. In explaining this, it must be borne in mind that the site of nasopharyngeal membrane often interposed between the head end of the gut tract and the primitive mouth cavity represents the point where the three primitive germ layers are closely applied to one another. Endodermal growths find their origin from the mucous membrane of the gut tract and Sessel's pocket, while ectodermal growths arise from the lining of the stomodeum and Luschka's pouch and mesodermic growths may arise between these two embryonic tissues.

With reference to my first point, I should like to show a short film in color illustrating examination of the nasopharynx in 2 patients. The first was a singer who, because of her

muscular control, needed no anesthetic for introduction of a self-retaining palate retractor and a large laryngeal mirror for the photographing. The second was a lad aged 19 with a nasal fibroma, whose soft palate had been cut to facilitate removal of the mass. A remnant of the mass appears in the left posterior choana.

Regular Meeting, Dec. 16, 1942

GEORGE L. WHELAN, M.D., *Recorder*

Basal Cell Lesions of the Nose and Lip: Relation to Roentgen Treatment and Plastic Surgery. DR. WARREN B. DAVIS.

Basal cell lesions should interest the rhinologist because of the frequency of their appearance about the nose, the upper lip and the cheek. Study of these tumors is not monotonous, for the etiologic factors are varied and the response of individual lesions to the different forms of therapy is far from uniform. They include, in addition to hyperkeratoses and many flattened, irregular, nodular masses, the so-called rodent ulcers. Most pathologists regard these tumors as growths derived from the lowermost, or basal, layers of the epidermis, for which reason they seldom show any tendency to keratinization or to pearl formation. Sections of such tumors show a complexity of downward growing strands, all reaching to about the same level, sharply outlined against the stroma and showing little inclination to strew cells into the irregular crevices of that tissue. This morphologic character is doubtless an expression of their relatively benign type of growth and the rarity of any tendency to metastasize. Later, however, necrosis and ulceration are common, and the tumor often presents itself as a slowly advancing, ragged, shallow ulcer, with a very thin wall of tumor tissue.

Etiologically, basal cell epithelioma may start in some chronic growth or change in the skin; after an acute injury; after such exposure to strong light or to harsh winds and weather as the many farmers and sailors with the tumor experience; after repeated irradiations, such as are given for acne, or sometimes apparently spontaneously, as a superficial red or pearly spot. Chronicity is usually an outstanding characteristic, but umbilication or ulceration may occur, either early or late. In the occasional case in which there appears to be metastasis to regional lymph nodes or to some distant part of the body, careful study will show that a squamous cell carcinoma has developed at the margin of the basal cell growth and that the "metastatic" lesion is of squamous, rather than of basal, cell origin.

Incipient and early basal cell growths can be destroyed by application of caustics, cauterization, fulguration, desiccation or treatment with radium or roentgen rays. To be safe the destruction must be complete. The defects resulting from the use of some of these methods are apt to leave more scarring than does a clean excision. It is probable that both radium and roentgen rays are more efficient than caustics. Many cures have been secured with each method of treatment. Recurrences, also, not infrequently follow each method of treatment. The recurrences are probably in direct proportion to the completeness of removal of the lesion.

The fate of the patient with a basal cell lesion rests largely on the judgment of the physician in charge during the early stages of the growth and his mastery of the method of treatment he first elects to employ. Some of the recurrences occur in patients who have been classified as cured for more than five years. In cases of early growth and in cases of recurrence in which the involvement and destruction have not been so extensive as to make complete excision feasible, I prefer excision with the cold knife and such plastic repair as is adaptable to the situation.

I have no statistics on the percentage of basal cell lesions which have been treated by the various methods, but my personal impression is that in the vast majority of cases roentgen ray treatments have at some time been used. The patients referred to me for plastic repair have usually had one or more recurrences, with repeated treatments. Often there have been considerable destruction of tissue and much fibrosis surrounding the area. The fibrosis is a definite handicap in plastic repair because of the decreased blood supply. While the eradication of the basal cell area is of course the primary consideration, the ultimate cosmetic appearance deserves careful consideration because of the psychologic and economic results. Thus, when it is found that the cells are not responding to radiation therapy as satisfactorily as might be expected, would it not be wiser, when the location of the lesion is favorable, to utilize surgical excision and plastic repair while the surrounding tissues are not too fibrosed and still have an adequate blood supply?

In cases in which there are wide and deep ulcerations and the growth tends to recur soon after supposedly adequate therapy, plastic reconstruction is not feasible. The use of a suitable prosthesis minimizes the patient's mental depression and permits social contacts.

Summary.—1. Basal cell lesions are characterized by their chronicity, slow local progress and absence of metastasis to local or distant areas.

2. The most important point in treatment is completeness of removal in the primary attempt, whatever method is employed.

3. In cases of early and moderately advanced lesions, excision and plastic repair offer good chances for cure, with minimum disfigurement.

4. In cases of late and repeated recurrences, with considerable destruction of tissue, the use of a prosthesis may be preferred to a plastic procedure.

DISCUSSION

DR. JOHN T. FARRELL JR. (by invitation): Dr. Davis' presentation of this important subject is timely. From the radiologic point of view, I concur in what he says regarding the importance of early diagnosis and wide destruction of these tumors. In my experience, each basal cell carcinoma is an entity of its own, and it is impossible to tell with certainty how any particular one is going to behave. The failures of roentgen treatment have probably been due to inadequate therapy. Widmann, who has had a vast experience, emphasizes that in radiation therapy a massive dose should be given when the lesion first comes under treatment and that an area of sound skin about the tumor should also be included in the treated field. Dr. Davis has pointed out that the objective in surgical intervention is similar—wide excision of the tumor, including some of the normal surrounding tissue.

The management of recurrence of basal cell tumors is difficult and unsatisfactory from the radiologic standpoint. In some instances squamous cell changes develop in the margins of a persistent basal cell lesion or in the margins of an area from which a basal cell growth has been excised or destroyed. Probably the best method of handling such a development is to treat it as one would a growth which has not received previous treatment.

The outcome is always uncertain, and frequently disappointing.

As Dr. Davis has said, and in my opinion this cannot be emphasized too much, the important factors in the treatment of basal cell cancer are early diagnosis and early radical treatment, regardless of the method of therapy employed.

DR. GEORGE M. DORRANCE: I wish to congratulate Dr. Davis on his presentation. No one can argue with him that excision is not a generally accepted method of treatment of basal cell carcinoma. There are times when roentgen ray or radium treatment should be used to supplement the surgical procedure.

Unfortunately, all carcinomas cannot be definitely classified as the basal cell or the squamous cell type. Many are of a mixed variety—some 10 per cent are "basosquamous." About 1 per cent are indeterminate in type. The latter, however, are usually so classified because the pathologist has been given a poor biopsy specimen.

I wish to congratulate Dr. Davis on his success in making a complete nose from a forehead flap. Surgeons doing this type of repair can appreciate the use of a skin graft in lining the flap, as well as the technic necessary to accomplish such a perfect result. I have not had Dr. Davis' success with cheek flaps for reconstruction of the upper lip, and I now rarely employ the method.

Role of Rhinoplasty in Restoration of Nasal Function. DR. ROMEO A. LUONGO.

Many rhinologists have not given enough thought to the importance of rhinoplasty as a part of their armamentarium. This is unfortunate, inasmuch as a large number of patients with rhinologic disorders cannot be completely relieved without supplemental surgical treatment of the external deformity. For instance, in the case of recent nasal fracture, the rhinologist corrects not only the deformities of the septum but those of the external framework. With old unreduced fractures, however, his corrective procedures are usually limited to the septum. Why not correct the external nasal defect as well? By so doing, not only will the external appearance of the nose be improved, but, as I shall endeavor to demonstrate, better physiologic function will be restored. Indeed, in many cases it is questionable whether there can be complete restoration of function without plastic measures.

One usually is concerned mainly with securing an adequate airway by various operations on the septum and turbinates. Many of these operations, while technically perfect, are only relatively successful from the functional standpoint because the proper distribution of air

currents, which is as important to olfaction and air conditioning as the provision of a free airway, has not been taken into consideration.

Inspired air does not follow a straight course from the nostrils to the choanae, but pursues a wide parabolic curve. Along the under surface of the dorsum of the nose, the nares give shape to this stream of air, and the constricted vestibule directs it upward along the olfactory sulcus. The olfactory sulcus is a channel-like space, extending from the vestibule to the pars olfactoria (Schaeffer). It is bounded anteriorly by rounded, or arched, confluence of the nasal walls and posteriorly by the agger nasi. The importance of proper configuration and position of the structures entering into the formation of this pathway in their relation to proper distribution of air is thus apparent. Hump, saddle and scoliotic deformities of the nose definitely affect this olfactory sulcus.

The turbinates, on the other hand, have little influence on the stream of inspired air. The anterior tip of the middle turbinate remains lateral to this current, practically avoiding it. Likewise, the anterior tip of the inferior turbinate lies considerably behind the channel of inspired air, and the body of the turbinate remains lateral even to the minor eddies which flow through the inferior meatus.

A misdirected current, either too hot or too cold, if concentrated on a circumscribed area, will evaporate the liquid contents of the lining, thicken the mucous secretions and impair ciliary movement, with consequent drying and crusting of the membrane and irritation of sensitive nerve endings resulting in intractable headache, neuralgia, frequent colds, anosmia, parosmia, etc. Even transient drying, according to Proetz, may incapacitate the cilia permanently. Likewise, too little air will fail to make an impression on the olfactory receptors, with resultant impairment of the olfactory sense.

As I have mentioned before, the shape, position and direction of the anterior nares also influence the course and distribution of the air currents. Deformities about this region are prevalent. McKenzie reported that 75 per cent of the men inducted into the armed forces have obstructions about the nasal entrances.

These deformities frequently escape recognition because the nasal speculum obscures or distorts them. The method of examination which I have found most satisfactory is as follows:

1. First, inspect the nose and observe the symmetry and contour of the tip, the patency of the nares, the site and shape of the alae, the size and shape of the columella and the position of the caudal end of the septum.

2. Elevate the tip with the finger. Note the condition of the cartilaginous septum. Note also whether or not breathing is improved by this elevation. If so, attachment of the tip at a higher level will afford relief and often eliminate unnecessary intranasal operation. The following case, which recently came under my observation, is illustrative.

Mr. S. G., aged 45, whose chief complaint was impaired nasal breathing, gave a history of four intranasal operations over a period of twenty years, all without material improvement. The first was a submucous resection; the second, an intranasal ethmoidectomy, and the third, a turbinectomy. Not until the tip was elevated, by attachment of the columella to the septum at a higher level, were his sympoms of obstruction relieved.

3. Next, with the thumb and the index finger in the nostrils, palpate the columella for movability, infiltration, stiffness, etc. Feel the connecting membrane between the septum and the columella; feel the posterior ends of the mesial crura. Note the thickness of the septum and its relationship to the vomerian ridge. Try to feel the nasal spine, and note its size and position.

4. Now, with a rake retractor, gently evert each ala and observe the condition of the upper lateral cartilages. Note whether they are dislocated, hypertrophied or misshapen. Also, note the angle that these cartilages form with the septum. A trained eye can easily recognize all these malformations. I use the speculum only after the inspection and the digital examination.

Rhinoplasty has recently reached such a high level of efficiency that it offers a splendid field for research and for further developments in rhinologic surgery.

DISCUSSION

DR. SAMUEL FOMON, New York: I have enjoyed Dr. Luongo's excellent talk and am entirely in accord with his views.

As an instructor, I have had occasions to talk with many nose and throat specialists regarding the place of rhinoplasty in their practice, and all have agreed on its importance. Despite this unanimity of opinion, few men have incorporated it in their practice. I assume that the reasons are twofold.

1. A fallacious belief that the rhinoplastic operation has a cosmetic objective. As a matter of fact, when cosmetic improvement is the sole reason for operation, the case had better be left alone, for cosmesis is a matter of taste, over which the surgeon has no control, and the results usually leave both physician and patient dissatisfied. Indeed, a person with a nose of reasonably normal appearance who seeks a nasal operation for the sole purpose of being made more beautiful than his neighbor needs a psychiatrist rather than a rhinoplastic surgeon. There is, however, the sensitive person with a noticeable deformity who desires its repair not to be made more beautiful but simply to be made less conspicuous. Such patients, I believe, deserve attention. Rhinoplasty, then, is indicated to obtain functional improvement and as a psychotherapeutic measure.

2. The belief that the operation is hazardous. This impression probably springs from the poor results obtained by inadequately trained surgeons. If the patient is carefully selected and the operator properly trained, rhinoplasty is a satisfying kind of surgery and complications are negligible, since one operates only on healthy tissues and on patients who are considered excellent risks.

The difference between the plastic surgeon and the otolaryngologist is that one sees the nose as an ornament and the other as an organ.

I believe that with otolaryngologic operations standardized, new horizons must be sought and that rhinoplasty should be incorporated as a part of the specialist's armamentarium. There is, however, a type of rhinologist who had best leave rhinoplasty alone.

DR. SAMUEL COHEN: Dr. Luongo's point that many persons with nasal obstruction can obtain proper relief only through a plastic operation on the nose is well taken.

Contrary to Dr. Fomon's experience, most of my patients who come for nasal correction do so because they believe their noses do not suit their faces. I usually agree with them. I think of nasal deformities as of four types.

The Long Nose.—The nostrils point downward and backward, with the tip of the nose pointing downward, instead of anteriorly. For years I have asked my postgraduate students to demonstrate on themselves how much improvement can occur when they elevate the tip of the nose with the finger. With this type of nose, the mere removal of a triangular wedge of the septum low down, with the base anterior, followed by proper suturing, not only shortens the nose but improves the breathing.

The Twisted Nose.—It is self evident that a nose which has a twisted appearance has twisted component parts as well. Correction of any one part, such as the nasal septum, will do little, if anything, to correct the nasal contour. The number of submucous resections done on noses of this type runs into the thousands yearly. I wish to stress, therefore, that a twisted nose often needs correction both of the septum and of the contour.

The Dislocated Septal Cartilage.—The septal cartilage is dislocated from the upper border of the columella, from the vomer and from its proper position between the split lower edge of the upper lateral cartilages and the lateral plates of the lower lateral cartilages. This produces a typical deformity of the lower part of the nose. The nasal tip is usually flattened and pushed over to the one side. Breathing is frequently impaired. In adults this condition is often corrected by the simple procedure of submucous resection of the dislocated piece of septal cartilage. The end result is improvement in breathing, but not in the nasal deformity.

It is only when the pathoanatomic changes associated with this deformity are fully understood and corrected that a good result can be obtained. Operations for its correction have been put forward under various names, but since the complete pathoanatomic picture has not been taken into consideration, the results are usually far from good.

The dislocated septal cartilage is frequent in children, with resultant poor nasal breathing and nasal deformity, a condition which makes the child conspicuous and calls urgently for proper correction.

I first described my technic for correction of this defect in 1928 (Lower Nasal Deflection, ARCH. OTOLARYNG. **8**:399 [Oct.] 1928), and have reported improvements in the technic in successive articles.

Postplastic Nasal Deformities.—After plastic repair of the nose, owing to infection or to poor technic, complications may arise which make the patient worse, instead of better. Such complications most frequently occur at the hands of the beginner, but can happen to any one. I have seen new postoperative deformities of the nasal tip ranging from a defect producing slight difficulty in breathing to complete vestibular and nasal streets. Dr. H. Miller showed me a case in which he had to reconstruct a vestibular lining to insure a slight breathing space.

In order to avoid deformities of the nasal tip, it is important to beware of removing parts of the lower lateral cartilages and the lining. Contractures of any kind can occur. In a recently published paper on complications of rhinoplastic procedures (*Dis. Eye, Ear & Throat* **2**:235 [Aug.] 1942), I described some of these complications which had occurred in my practice and in that of about fifty other plastic surgeons in this country.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

G. HENRY MUNDT, M.D., *President*

Regular Meeting, Dec. 7, 1942

Cricopharyngeus Muscle: A Roentgenologic Demonstration. DR. FREDERIC E. TEMPLETON (by invitation).

Anaerobes in Cranial Osteomyelitis. DR. THOMAS C. GALLOWAY, Evanston, Ill.

The usual explanation of cranial osteomyelitis on the basis of thrombophlebitis does not state (1) why involvement of the maxilla and other compact bone may be so serious or (2) why fulminating infection is more likely to follow minor intervention. The characteristics of such an invasion might be accounted for by the spread of microaerophilic streptococci along the periosteum, as Meleney demonstrated them burrowing along fascial planes; a minor operative procedure would make a covered focus in which anaerobic organisms could grow well. When, with proper technic, I have looked carefully for these organisms in cultures of material from the depth of the lesion or the advancing border of the disease, I have nearly always found them. Nine cases are reported.

Anaerobes seem to be the primary invaders. That they are the important organisms is supported by the striking response of the infection to careful treatment with activated zinc peroxide, in contrast to the course of similar infections which were not treated with this agent. Severe pain, tenderness, foul odor and induration are characteristic. Anaerobes probably have an important role also in resistant infections of soft tissues about the head, such as Ludwig's angina.

DISCUSSION

DR. SHERMAN L. SHAPIRO: This paper is an important contribution, for it emphasizes that the anatomic explanation of Furstenberg and others does not account entirely for these infections. All have observed that infections of the frontal bone and the superior maxilla do not always respond to therapy in the same way, regardless of their looking alike. It is possible that Dr. Galloway has the solution to the problem. Recent military literature has mentioned that anaerobes have a predilection for bone. The spread along the periosteum may supply the reason that the clinical behavior in cases of cranial osteomyelitis is so different.

My own experience with zinc peroxide is slight. I tried it in a case which I reported last year. The patient recovered with conservative surgical treatment. Zinc peroxide did not achieve a cure, perhaps because of the inaccessibility of the infected bone in that case. In a second case of stubborn infection of the antrum I was much impressed by the results with the drug; the discharge was noticeably less, the odor disappeared and a cure was obtained.

I think Mosher emphasized the importance of Staphylococcus aureus in osteomyelitis. Is it Dr. Galloway's opinion that this organism lacks ability to produce damage, or does he believe that it can itself produce osteomyelitis?

DR. GORDON H. SCOTT: Dr. Galloway suggests that infection in the cranial bones travels by way of the contiguous veins, rather than through the diploic spaces. This does not explain the discrepancy in the speed at which infection travels in compact and in diploic bone.

DR. DELBERT K. JUDD: I believe this subject will prove of great importance in the therapy not only of osteomyelitis but of other infections of the nose and throat. At least one cannot neglect to culture for anaerobes, in spite of certain difficulties in technic. I wonder whether such persistent and frequent culturing of material from a great many types of infection might not eventually yield anaerobes.

Dr. Galloway spoke of the cultures made in the 7 cases of osteomyelitis which Dr. Mosher and myself reported (*Laryngoscope* **43**:153 [March] 1933). It is true that the cultures were all positive for the staphylococcus, with the additional presence of streptococci in 1 case, but now it seems that these organisms may have been of secondary importance. Anaerobic streptococci may have been present, since cultures for anaerobic organisms either were not made at all or were not made frequently enough to be of value. I do not remember that the presence or absence of anaerobes was considered. With respect to spread of the disease through the bones of the calvarium, it is established that the process of retrograde thrombosis and thrombophlebitis plays an important role. In all the 7 cases, however, the inner table of the frontal bone or other involved bones of the calvarium was removed because of the frequency of extradural abscess with osteomyelitis. Furstenberg has shown that the infection spreads by way of the dura or the endosteum. As the endosteum is stripped away from the inner table, the bone is robbed of its chief source of blood supply, with resulting necrosis and sequestration.

Dr. Howard C. Ballenger, Winnetka, Ill.: I wonder whether Dr. Galloway has tried zinc peroxide in treatment of persistent maxillary sinusitis following extraction of an infected tooth. Many of the bacteria in the sinus are anaerobes.

Dr. Edwin J. Blonder: Is zinc peroxide irritating to the eyes, and, if so, is any protection given the eyes when the drug is used in the vicinity?

Dr. Glenn J. Greenwood: I was interested in noting the relative times at which the cultures were taken in the course of the infection. Altemeier, reporting on the bacterial content of war wounds, remarked that anaerobes present the first week have, by the third week, been replaced by secondary pyogenic invaders. He expressed the belief that careful primary débridement is one of the most effective prophylactic and therapeutic measures employed in treating the anaerobic component of the infection.

Dr. Thomas C. Galloway, Evanston, Ill.: I do not think Dr. Shapiro's question concerning the role of staphylococci can be answered with finality. In hematogenous osteomyelitis this organism seems able to do great damage by itself; yet in cranial involvement it is more likely to be a secondary invader. Perhaps, however, it is responsible for the fulminating phase.

As to Dr. Scott's question regarding the method of spread of osteomyelitis, Mosher holds that the diploic venous spaces are most important, and Furstenberg, the dural veins. I should expect anaerobes to travel not in the vessels but along avascular fascial planes—for instance, under the periosteum of flat bones. Perhaps, again, when venous spaces are reached, the secondary staphylococcic invaders become important.

Zinc peroxide has not seemed irritating to the eyes when used near them, although keeping the lids closed has kept it out rather well.

In just such a case of maxillary sinusitis as Dr. Ballenger mentioned, in which anaerobic streptococci were obtained in cultures from an antrum with disease secondary to dental infection, zinc peroxide was effective.

Débridement, as mentioned by Dr. Greenwood, should be effective, both by removal of necrotic infected tissue and by exposure of any infection to aerobic conditions.

Post-Traumatic Rhinoplasty: Secondary Correction of Septal Deviations and External Deformities of the Nose. Dr. Samuel Salinger.

Review of a series of more than 1,000 cases of operative correction of the septum and nose reveals that a large proportion of deviations of the septum are to some degree the result of previous trauma. While it is generally believed that deviations of the septum are the result of errors in development, usually a disproportionate rate of growth between the septal structures and the bony cage surrounding the nose, careful analysis of many cases shows irregularities that could have been caused only by injury. Nearly all rhinologists have discovered that sooner or later the classic submucous operations of Killian and Freer are inadequate to cope with the situation; individual improvisations have crept into the technic, varying with the skill and ingenuity of the operator.

In many cases correction of the septal deflection must be linked with correction of the external deformity. Unless a twisted nasal bridge is straightened the submucous resection will often fail to give an adequate airway. Various procedures, such as the Metzenbaum technic, partial resection of the septum, hacking of the septal cartilage or removal of narrow strips to permit straightening of a curve, must be employed in order to preserve an intact septum and prevent the collapse which frequently follows complete removal of the obstruction.

Various types of incision, designed to fit the individual case, are shown, together with the procedure for straightening a deviation of the nasal bridge. As a rule the combined technic, rather than separation of the procedure into two steps, is preferred. This makes for better appraisal of the amount of septal tissue that must be sacrificed and tends to conservation of a greater amount than is usually possible if the septum is resected without regard for the external deformity.

DISCUSSION

DR. JOHN F. DELPH: I should like to ask whether it is advisable to attempt to correct the nose immediately after injury, or to wait until the landmarks can be seen, after the initial edema has subsided and the soft tissue has been repaired.

DR. WALTER H. THEOBALD: I wish Dr. Salinger would state his opinion as to the time after injury and the age of the patient at which operation on a deviated septum is advisable. He showed a picture of a child whom he treated for three or four years prior to operation. Opinions vary as to whether operation should be performed at all before the age of puberty, or even before the age of 18 years. I have always discouraged operative procedures on the nose until the patient is 19 or 20 years of age. However, some men operate prior to that age, to give an airway, when the deviation is severe and then complete the operation on the septum later.

DR. GEORGE H. WOODRUFF, Joliet, Ill.: I wish Dr. Salinger would describe his procedure in a case in which he sees the patient within a week or ten days after a crushing injury to the nose, which would result in a traumatic saddle nose later.

DR. SAMUEL SALINGER: In reply to Dr. Delph, a delay of twenty-four or forty-eight hours is likely to result in infection, which is the chief factor in interference with smooth healing. In the early correction of nasal injuries I am not worried about initial edema; it is not bothersome. Most lacerations if properly cleansed and sutured early and accurately will heal with practically no scarring. When suturing is inaccurate or the wrong suture materials are employed, scarring will result and necessitate a secondary correction. Of course, there are cases of massive injury and shock in which it is inadvisable to do anything but control hemorrhage and institute local antiseptic measures; yet if the wound is carefully sutured and the structures replaced within twelve to twenty-four hours the good results of early care are often duplicated. In a case of injury from flying glass two months ago, the nose and septum were completely cut across. I saw the patient within two hours of the accident, before any reaction had set in, and was able to sew up the septum, columella, nasal vestibule and external portion of the nose so that today one can barely see the site of the laceration.

In reply to Dr. Theobald, I should not do a submucous resection prior to the age of 16 or 17 years. However, this does not mean that one should not operate on a septum before this age. In the case of the child of 11, I removed only the lower end of the septal cartilage; the rest of the cartilage was infracted with the vomer and pushed back to the midline. Sometimes in children the lower end of the septum is buckled so as to block both nares. In such a case relief is needed, but not necessarily a submucous resection. One must contrive so to manipulate the cartilage and supporting bone, if necessary, as to provide breathing space without sacrificing the growing structure.

With regard to prevention of a saddle depression after trauma, every attempt should be made to elevate and retain the depressed cartilage and bone. Despite all precautions, however, deformity will result from the absorption of small fragments and scar contraction. The damage is not irremediable if one is prepared to insert a cartilage implant later. I have done this on several occasions, with excellent cosmetic results. In a case in which a compound fracture directly across the lower ends of the nasal bones resulted in depression, the defect was subsequently filled in with a single cartilage isograft.

Book Reviews

Diseases of the Nose, Throat and Ear: Medical and Surgical. By William Lincoln Ballenger, M.D., F.A.C.S., Late Professor and Head of the Department of Otology, Rhinology and Laryngology, University of Illinois College of Medicine, and Howard Charles Ballenger, M.D., F.A.C.S., Associate Professor of Otolaryngology, Northwestern University Medical School. Eighth edition, thoroughly revised. Price $12. Pp. 975, with 604 engravings and 27 plates. Philadelphia: Lea & Febiger, 1943.

This standard textbook has now reached an eighth edition, retaining all the excellent features of the previous edition, but with some of the material rearranged, rewritten and enlarged.

The junior author has preserved that indefinable balance of the former editions, and he should be congratulated on his clear setting forth of the fundamental, well grounded principles and procedures of good otolaryngologic practice.

The following specialists have contributed to this volume: Dr. B. T. King describes his operation for bilateral paralysis of the recurrent laryngeal nerve and Dr. J. D. Kelly his technic of arytenoidectomy for the same condition; Dr. C. L. Jackson has revised the chapter on esophagoscopy; Dr. Alfred Lewy discusses the labyrinth with respect to physiology, functional tests and inflammatory diseases, and Dr. J. J. Ballenger writes on the paralyses and associated neuroses of the larynx. Many other operative procedures are fully considered, among which is a comprehensive description of the technic of the fenestration operation of Dr. Julius Lempert, with full credit to all the noted otologists of Europe and America who have contributed to this operation.

Numerous diagrams, schemas, plates, engravings, photographs and photomicrographs enhance the value of the well written text material, so that the entire theme is spirited and dynamic. Interest and concentration are fostered, and many readers may find their retentive powers increased by the lucidity of the message which the junior author has sincerely tried to impart.

The entire subject is divided into five parts: The Nose and Accessory Sinuses; The Pharynx and Fauces; Diseases of the Larynx; The Ear, and Bronchoscopy.

Accepted procedures in medical and surgical treatment are outlined, and the discussion of therapy is complete and detailed from all possible angles.

This reviewer has remarked on the high grade instructional value of the preceding edition, and he wishes to make the same comment on the present one, particularly with regard to the following features: the classic description of the deformities of the nasal septum and their surgical correction; the concise description of thrombosis; the chapter on the singing voice, which gives valuable information and throws new light on respiratory efficiency, control of the expiratory air current and relationship of the mechanics of the chest and abdomen to the richness, quality and power of the voice, and the crystal clear description of the treatment of diseases of the eustachian tubes, the last consideration being worthy of glowing comment.

This textbook is recommended to every otolaryngologist, physician in general practice, medical student and graduate student in otolaryngology and to those taking residencies in the specialties of otolaryngology and ophthalmology.

Dr. Howard C. Ballenger has contributed much to American otolaryngology and to the teaching of this specialty, and now his efforts will aid no little the military authorities of the United Nations. Thank you, Dr. Ballenger.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Claude C. Cody, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.

American Academy of Ophthalmology and Otolaryngology

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
President-Elect: Dr. Lawrence T. Post, 508 N. Grand Blvd. St. Louis, Mo.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Place: Chicago. Time: Oct. 10-13, 1943.

American Broncho-Esophagological Association

President: Dr. Robert L. Moorhead, 125 Ramsen St., Brooklyn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.

American Laryngological Association

President: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James G. Dwyer, 375 Park Ave., New York.
President-Elect: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.

SECTIONS:

Eastern.—Chairman: Dr. Edward J. Whalen, 750 Main St., Hartford, Conn.
Southern.—Chairman: Dr. Francis B. Blackmar, Swift Kyle Bldg., Columbus, Ga.
Middle.—Chairman: Dr. Voss Harrell, Hofman Bldg., Detroit.
Western.—Chairman: Dr. Irving M. Lupton, Medical Arts Bldg., Portland, Ore.

American Otological Society

President: Dr. Wesley C. Bowers, 17 E. 61st St., New York.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.